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SYNDROME OF MENINGEAL FIBROBLASTOMA ARISING FROM LESSER WING OF SPHENOID BONE

ROBERT A. GROFF, M.D.

PHILADELPHIA

In a previous study [1] was described a definite clinical picture noted in patients with a meningeal fibroblastoma arising from the lesser wing of the sphenoid bone. That investigation was based on four cases, and all the characteristics of this tumor were not included. Since that communication was published five other patients with a tumor in the same location and manifesting additional signs and symptoms have come under observation. These five cases are reported here in detail, and all nine are discussed in order to clarify the syndrome.

The signs presented by such a tumor are caused by involvement of the structures in the region of the lesser wing of the sphenoid bone (fig. 1). The most significant of these structures are the optic nerve and chiasm, which lie medially and slightly posterior to the lesser wing. The third, or oculomotor, nerve pierces the dura just lateral to the chiasm, passes through the cavernous sinus and enters the orbit through the superior orbital fissure. Coursing in the same general direction but passing through the dura more posteriorly are, successively, the trochlear and the abducens nerve. The abducens nerve is shielded by the ophthalmic nerve as all three pass through the cavernous sinus and the superior orbital fissure to enter the orbit. These nerves therefore may be subjected to pressure by a tumor arising from the lesser wing of the sphenoid bone when it has a paramedian position in the middle fossa. If the tumor extends forward, the olfactory nerve, which is anterior and medial to the lesser wing, may be compressed. Besides these cranial nerves, the pituitary gland, the uncus and the internal carotid artery and its immediate branches are in close relationship with the sphenoid ridge and may be subjected to pressure by the tumor. It is these various structures which are of chief concern in a consideration of tumors in this location.

From the Neurosurgical Clinic and Laboratory of the Hospital of the University of Pennsylvania and the Graduate Hospital of the University of Pennsylvania.

1. Alpers, B. J., and Groff, R. A.: Parasellar Tumors: Meningeal Fibroblastomas Arising from the Sphenoid Ridge, Arch. Neurol. & Psychiat. **31**:713 (April) 1934.

The clinical syndrome of a tumor of the lesser wing of the sphenoid bone, as previously stated, consists of primary atrophy of the optic nerve or the Kennedy syndrome; defects in the visual fields, which usually take the form of complete homonymous hemianopia but which may be quadrantic; paralysis of the third nerve, and unilateral impairment of the olfactory nerve. Unilateral exophthalmos is present in some cases. Additional symptoms, such as motor paralysis (monoplegia), impairment of memory and disturbances of the pituitary body, are observed. The roentgenograms, although frequently revealing no abnormality, show changes in the sella turcica—a "top normal" size, slight erosion of the dorsum and of the anterior clinoid processes and unilateral deformation of the sella in some instances.

The following case reports will aid in the clarification of the syndrome.

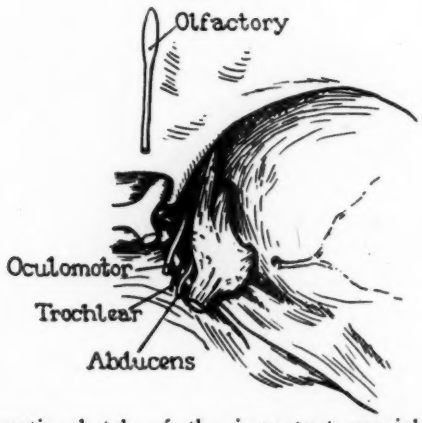

Fig. 1 —Diagrammatic sketch of the important cranial nerves close to the lesser wing of the sphenoid bone.

REPORT OF CASES

CASE 1.—*Small tumor of left sphenoid ridge encircling left optic nerve. Symptoms for three years. Left eye, exophthalmos; blind. Right eye, slight defect in lower temporal quadrant; vision, 6/5. Right disk normal; left disk, advanced primary atrophy. Impaired corneal reflex on left. Partial palsy of left third nerve. Pituitary fossa, 9 by 11 mm. Erosion of lesser wing of sphenoid bone on left. Operation and complete removal.*

History.—H. E., a man aged 42, was referred to the service of Dr. Charles H. Frazier in the University Hospital by Dr. T. H. Farrell, of Rochester, N. Y. This case has been previously reported by Dr. Frazier.[2] The patient had been well until August 1931, when an intermittent aching pain developed in the left arm, which lasted for five months. In December he noted a steady aching pain over the left malar bone and ala of the nose, which was accentuated by a sudden jar. This pain suddenly stopped in April 1932. The patient was practically well

2. Frazier, C. H.: A Review of Clinical and Pathological Para-Hypophyseal Lesions, Surg., Gynec. & Obst., to be published.

except for a period of loss of energy and of easy fatigability and a loss of 20 pounds (9.1 Kg.) in weight from October 1932 to July 1933, when he began to have blurring of vision in the left eye. He saw double and suffered from aching pain in the left eyeball. The pain along the left side of the nose and cheek returned. At the same time vision in the left eye began to fail, and the eye began to swell. By December 1933 there was definite prominence of the left eye. At that time what was thought to be an osteoma of the lateral wall of the left orbit was removed at another hospital. The patient experienced no relief. In April 1934 the left eye became blind. These symptoms continued until he was admitted to the hospital.

Examination.—There was definite exophthalmos of the left eye (right, 16.5 mm.; left 18.5 mm.). The lid of the left eye drooped; the pupils were unequal, the left being larger than the right; the right pupil reacted to direct but not to indirect light; the left pupil reacted to indirect but not to direct light; both pupils reacted in accommodation and convergence. The rotations of the right eye were full. There was slight impairment of movement in the left eye in all directions. Examination of the fundus revealed a normal right disk and a gray-white atrophy with fuzzy disk margins in the left eye. The vessels were within normal limits. The left eye was totally blind, and the right eye showed a slight defect in the lower quadrant of the field on the temporal side. The visual acuity was 6/5 in the right eye. Roentgenographic studies showed a definite erosion of the lesser wing of the sphenoid ridge on the left side. The pituitary fossa measured 9 by 11 mm.

Operation.—In November 1934 Dr. C. H. Frazier made a left frontotemporal osteoplastic flap. Exploration of the left sphenoid ridge revealed a tumor on the mesial aspect of the lesser wing. The tumor began just above the lesser wing of the sphenoid ridge and extended up to and completely surrounded the left optic nerve. It extended into the middle fossa, terminating over the second division of the gasserian ganglion. The tumor was removed completely.

Pathologic Examination.—The pathologic examination was made by Dr. B. J. Alpers. The tumor was a meningeal fibroblastoma, composed in large part of collagen fibers placed in parallel rows with fibroblasts among them. In other parts the tumor was more cellular, the cells containing oval pyknotic nuclei, which were arranged in rows. The growth was very vascular and fibrous.

Comment.—The diagnosis in this case was uncertain because of the unilateral exophthalmos. The long history, extending over a number of years, was confusing until vision centered on the hyperostosis of the lateral wall of the left orbit as the cause of the symptoms. The clinical findings of complete blindness of the left eye, a defect in the lower quadrant of the right field, secondary atrophy of the left optic nerve, partial palsy of the third nerve and erosion of the sphenoid bone agreed with the syndrome of tumor of the lesser sphenoid wing and established the diagnosis.

Unilateral exophthalmos has been observed in a patient with a large meningeal fibroblastoma of both wings of the sphenoid bone but not in cases of tumor confined to the lesser wing. It was explained by the hyperostosis of the lateral wall of the orbit. This sign must be included as a part of the syndrome.

Subjective and objective sensory disturbances in the region supplied by the trigeminal nerve were not included in the earlier description of cases of this type of tumor. In this case the disturbance was characterized by paresthesias in the area supplied by the first and second divisions. The left corneal reflex was diminished, but there were no objective sensory findings in the face. The fact that the tumor rested on those nerves accounted for the disturbance. This sign must be incorporated in the syndrome and may accompany a tumor which projects into the middle fossa, pressing on the trigeminal nerve.

The following case, aside from presenting a characteristic picture, again emphasizes the signs of unilateral exophthalmos and paresthesias over the area of distribution of the trigeminal nerve accompanying such a tumor.

CASE 2.—*Tumor of lesser wing of left sphenoid bone. Headaches for twenty-five years; failing vision of left eye for five years. Complete blindness in left eye; 6/9 vision in right eye. Slight temporal hemianopia in right visual field. Secondary atrophy of optic nerves more marked on left. Disk margins blurred. Moderate exophthalmos of left eye. Paralysis of left third and sixth nerves. Pituitary fossa, 12 by 11 mm. A mass posterior to pituitary fossa on left with calcified periphery. Operation and subcapsular evacuation.*

History.—S. L., a woman aged 60, was referred by Dr. Jacob Greenwald and Dr. N. J. Davis, of Chesapeake, Md., to the service of Dr. C. H. Frazier in the University Hospital. There was a long history of headaches and a more recent history of failing vision in the left eye. When the patient was first admitted to the hospital, in June 1931, she had been suffering from headaches for twenty years. They occurred about twice a year and were generalized either on the right or on the left side. Occasionally they were accompanied with nausea and vomiting. In October 1930 she had an attack during which she complained of a terrific odor of old smoke and burning rags. In November 1930 a headache developed in the left frontotemporal region, which lasted for three months. Just before that she noticed failing vision in the left eye. Shortly after the headache began the left eye became practically blind, and the lid became paralyzed. The patient remained free from headaches until February 1931, when a similar headache on the left side occurred, which lasted for four months. Neurologic examination in June 1931 revealed complete left temporal hemianopia; primary atrophy of the optic nerves, more marked in the left eye; palsy of the third and sixth nerves, and paresis of the left fourth nerve. There was pain in the region of the first division of the left fifth nerve. An encephalogram was recommended, but the patient left the hospital before it was made. The interval history up to the time of the second admission in March 1935 disclosed frequent attacks of excruciating pain in the left side of the head, extending from the vertex into the left eye, and complete blindness of the left eye. During one attack of headache the patient lost consciousness for one hour.

Examination.—The neurologic findings in March 1935 were similar to those described on the previous admission. Exophthalmos had developed in the left eye, which was blind. The visual acuity of the right eye was 6/9, and the field showed a beginning temporal defect. There was secondary atrophy of the optic nerves, which was complete on the left. The disk margins were blurred. There were

paralysis of the left third and sixth nerves and paresthesias in the region supplied by the first division of the trigeminal nerve. The fourth cranial nerve was not affected. Roentgenograms of the skull, which were normal in 1931, showed a "top normal" sella turcica, measuring 12 by 11 mm. There was a circumscribed mass just posterior and to the left of the pituitary fossa. This mass had a calcified periphery and showed irregular deposits of calcium, which appeared to be in the wall.

Operation.—On March 26, 1935, Dr. C. H. Frazier turned back a bone flap in the left frontotemporal region. Exploration of the left sphenoid ridge disclosed a moderate-sized tumor, arising from the lesser wing. It projected into the middle fossa and lay close to the pituitary fossa. When an attempt was made to remove it, the mass pulsated, but fluid was not recovered when a needle was introduced. A subcapsular evacuation was accomplished.

Pathologic Examination.—Dr. B. J. Alpers examined all the fragments removed at operation. They were composed of a homogeneous mass of tissue which took an intense acid stain and had no structure. For the most part there was no visible cellular structure, except that in a few places fibroblasts were seen. In many parts of the tissue there were large cholesterol clefts, but there was nothing to demonstrate that the tumor was a cholesteatoma. Vascular spaces were a very unusual finding. Because of the indefinite pathologic picture demonstrated by the fragments received, this tumor remains unclassified.

Comment.—The patient presented a typical picture of tumor of the lesser sphenoid wing four years before verification. Operation was recommended at that time, but the patient refused to permit it. The neurologic examination at that time revealed primary atrophy of the optic nerves, more marked on the left; complete left temporal hemianopia; paralysis of the left third and sixth nerves; slight involvement of the fourth nerve, and involvement of the ophthalmic division of the fifth cranial nerve. Roentgenograms did not reveal any abnormality.

The examination made prior to operation, four years later, showed a progression of the clinical picture. The left eye was totally blind and moderately exophthalmic. The paralysis of the third and sixth cranial nerves was unchanged. No paresis of the oculomotor nerve could be detected. The subjective involvement of the area supplied by the ophthalmic division of the trigeminal nerve was more marked. The roentgenographic findings localized the tumor by demonstrating the presence of calcification. The sella turcica was of "top normal" size and there was partial demineralization of the posterior clinoid processes.

The headaches, variable as to location, were probably not caused by the tumor until they became localized in the left frontotemporal region and were the result of pressure on the trigeminal nerve. The unilateral exophthalmos was a late development and an important sign, as shown in the preceding case. The explanation for its development is not clear, unless it was due to interference with the return blood flow from the orbit by compression of the cavernous sinus. The paresthesias and pain in the area supplied by the ophthalmic division of the trigeminal

nerve were explained by pressure of the tumor on the nerve. Palsy of the sixth nerve on one side is not considered of focal value, as pointed out by Spiller.[3] However, its presence as part of the clinical picture of a tumor of the lesser wing is significant. It denotes a paramedian and somewhat posterior position of the tumor, which was the location of the lesion in this case.

The next case demonstrates all the characteristic roentgenographic findings seen in cases of tumor of the lesser wing.

CASE 3.—*Small tumor of flat type extending along whole length of sphenoid ridge and encircling right optic nerve. Increasing exophthalmos for one year. Early primary atrophy of right optic disk; normal left optic disk. Visual acuity in right eye, 6/9.5; in left eye, 6/12. Right visual field, concentric contraction; left, normal. Slight hypesthesia over first division of right fifth nerve. Spinal fluid pressure, 8 mm. of mercury. Definite erosion of walls of pituitary fossa, 15 by 12 mm. Marked increased density and new bone formation in region of right sphenoid ridge. Erosion of upper border of right optic foramen. Operation and incomplete removal.*

History.—R. A., a Negress aged 26, was referred to the service of Dr. F. C. Grant in the Graduate Hospital by Dr. T. H. Weisenburg, of Philadelphia. She had been well until one year before entrance, when she noticed beginning prominence of the right eye. The exophthalmos gradually increased, especially during the two months before admission. A few weeks before admission she noticed that the eyes became fatigued easily when she was doing close work, and transient black spots were seen.

Examination.—The right eye showed definite exophthalmos (right, 25 mm.; left, 16 mm.). The visual acuity was 6/12 on the right and 6/9.5 on the left. The left optic disk was normal; the right showed early primary atrophy. The visual fields were full on the left and concentrically contracted on the right. The area supplied by the first division of the right fifth nerve was definitely hyperesthetic. The spinal fluid pressure was 8 mm. of mercury. A roentgenogram of the skull showed marked deformity of the sella turcica with enlargement (15 by 12 mm.). The optic foramen showed definite encroachment on the right. There was evidence of erosion of the upper border of the sphenoid fissure, and increased density and new bone formation were noted about the sphenoid ridge on the right side. The posterior clinoid processes were atrophied and pointed.

Operation.—On Dec. 7, 1933, Dr. F. C. Grant performed a transfrontal craniotomy on the right side. Exploration of the right sphenoid ridge exposed a soft, reddish, flattened, encapsulated tumor. It occupied the entire length of the sphenoid ridge and embedded the right optic nerve. Most of the tumor lay in the middle fossa and was characteristic of the meningioma *en plaque*. It was incompletely removed.

Pathologic Examination.—The pathologic examination was made by Dr. B. J. Alpers. The architecture of the tumor was that of a typical meningeal fibroblastoma with whorl formation. The tumor was not particularly vascular.

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3. Spiller, W. G.: Bilateral Paralysis of the Facial and Abducent Nerves as Remote Effect of Tumor of the Brain, Ann. Surg. **101**:329, 1935.

Comment.—The history in this case was short, extending over a year, with rapid progression two months before admission, and was confined to exophthalmos of the left eye and minor visual disturbances.

The tumor was unusual because, unlike all the others, it was flat and small and covered the entire sphenoid ridge. It enveloped the right optic nerve and encroached on the optic foramen. This fact explains the roentgenographic findings of erosion of the optic foramen and lesser sphenoid wing. Part of the tumor compressed the first division of the trigeminal nerve, accounting for the hyperesthesia in that region.

This case demonstrates practically all the roentgenographic changes observed in cases of tumor of the lesser sphenoid wing—enlargement of the sella turcica, erosion of the lesser wing of the sphenoid bone and encroachment on the optic foramen. Although these manifestations are not always present, one or more of the triad, with the clinical picture, warrants the diagnosis of a lesion in that location. Aside from these findings the roentgenograms demonstrated hyperostosis of the bones about the sphenoid ridge. That is an unusual location for such a process.

Unilateral exophthalmos was present, as well as hyperesthesia in the region of the first division of the trigeminal nerve. Their occurrence emphasizes again that these signs are a part of the syndrome.

The difficulties in making a diagnosis when the syndrome is well advanced and the tumor is large are demonstrated in the following case.

CASE 4.—*Large tumor of lesser wing of sphenoid bone. Symptoms for nine years. Left pupil fixed to light and in convergence. Slight jerky nystagmoid movements on lateral gaze. Marked primary atrophy of optic nerves. Left eye, blind; right eye, complete temporal hemianopia with 6/22 vision. Spinal fluid-pressure, 140 mm. of water. Pituitary fossa, 13 mm. wide and very shallow. Encroachment on and obliteration of cisterna chiasmatis and upward displacement of third ventricle and anterior horns of lateral ventricles, as shown by encephalogram. Partial removal of large tumor of lesser wing of left sphenoid bone.*

History.— B. H., a woman aged 35, was referred to the service of Dr. C. H. Frazier in the University Hospital by Dr. G. Dorrance and Dr. W. G. Spiller, of Philadelphia. The rather long and periodic history was confined to the eyes. In 1925, following the birth of her third child, she noticed that vision in the left eye was dim and that both eyes tired easily. There was no progression of those symptoms until 1928, when she noticed advancing loss of vision in the left eye and for the first time loss of vision in the right eye. By March 1929 the patient was unable to read. However, following the birth of her fourth child she discovered a return of eyesight so that she could read. Vision in the left eye was less acute than that in the right. In April 1930 the vision in the left eye began to fail. In the winter of 1932 the sight in the right eye receded. The left eye became virtually blind. By the time of her admission to the hospital in December 1933 the left eye was totally blind, and there was little sight left in the right eye. There were no other complaints.

Examination.—The abnormal neurologic findings centered about the eyes. The pupils were regular and equal. The right pupil reacted to light and in convergence, but there was no consensual reflex. The left pupil did not react to light or in convergence, but the consensual reflex was present. Both disks showed waxy white primary atrophy. The left eye was blind; the right showed a complete temporal cut, with a visual acuity of 6/22. There were slight jerky nystagmoid movements on lateral gaze to the left and to the right. The spinal fluid pressure was 140 mm. of water. The pituitary fossa was 13 mm. wide and very shallow.

Encephalography.—The confusing clinical picture made encephalography necessary for more accurate localization and estimation of the size of the lesion. The unusual features presented in the encephalogram were absence of cortical pathways in the midline, encroachment on and obliteration of the cisterna chiasmatis and upward displacement of the third ventricle and of the anterior horns of the lateral ventricles.

Operation.—On Dec. 18, 1933, Dr. C. H. Frazier elevated a bone flap in the left temporofrontal region. The tumor was situated on the lesser wing of the sphenoid ridge, extending into the anterior and middle fossae as well as across the midline for a short distance. All the tumor that was in the anterior fossa was scalloped away. When an attempt was made to remove the portion of the tumor in the middle fossa, sharp bleeding was encountered from the capsular vessels; so complete removal was abandoned.

Pathologic Examination.—The pathologic report by Dr. B. J. Alpers stated that the tumor was a typical meningeal fibroblastoma. There were an unusual number of vessels present. A section of bone showed infiltration by tumor tissue that was not marked but fairly definite. There were small collections of fibroblastic cells in the bone spaces. Otherwise, the bone looked normal, little or no evidence of proliferation or destruction being detected.

Comment.—The diagnosis in this case was difficult to make because the neurologic picture was well advanced. The history of a beginning loss of vision in the left eye suggested a growth predominantly on the left side. In order to confirm that impression and to determine the size of the growth, an encephalogram was made. That examination did not lateralize the lesion but placed it in the midline in the suprasellar region. The operative exposure was therefore based on the clinical findings and the history. The tumor was large. It arose from the lesser sphenoid wing, extended into both the anterior and the middle fossa and over the optic nerves and chiasm. That location adequately explained the encephalographic pictures and accounted for the atrophy of both optic nerves, blindness of the left eye, complete temporal hemianopia of the right eye and deformity of the sella turcica.

The history was the longest in the nine cases recorded, extending over nine years. The symptoms were entirely referred to the eyes and were characterized by remissions and relapses. That peculiarity was undoubtedly influenced by the two pregnancies which occurred during the course of the disease. Before the birth of her last child the patient was unable to read, but following the pregnancy the vision improved

sufficiently so that she could read. Almost a year elapsed before failing vision was again noticed. The cause for that may have been either one of two processes or both. During pregnancy the pituitary gland enlarges, and the optic nerve and chiasm may have been compressed between the gland and the tumor. As the gland receded in size following the pregnancy, that pressure was relieved. On the other hand, the tumor may have become larger during pregnancy as a result of the increased blood supply, thus increasing the bulk of the tumor and the pressure on the optic nerves and the chiasm. With the termination of pregnancy the blood supply was reduced, with consequent reduction in the size of the tumor. It is probable that both factors played a part in the increase of symptoms during pregnancy and in the temporary improvement after delivery.

CASE 5.—*Moderate-sized tumor involving mesial aspect of right sphenoid ridge and extensive hyperostosis of right frontotemporal region and base. Temporal bulge for twenty years; symptoms of tumor for one year. Large bony swelling of right frontotemporal bone. Right exophthalmos. Right pupil dilated and fixed to light and in accommodation. Slight weakness of right rectus muscle, as well as limitation of upward and downward movements. Bilateral postpapillitic atrophy with distinct margins. Vision in left eye, 6/15; right eye, 6/60. Concentric contraction of right visual field. Pituitary fossa, 11 by 10 mm. Large area of hyperostosis of frontotemporal bones and base of skull on right. Operation: incomplete removal of involved bone and partial removal of tumor.*

History.—V. B., a woman aged 45, was referred to the service of Dr. C. H. Frazier in the University Hospital by Dr. S. L. Freeman, of Wilkes-Barre, Pa. She had first noticed a hard lump appearing on the right side of her forehead when she was 20. The swelling gradually increased in size over a period of years. Aside from the disfigurement, it gave no symptoms. One year before admission to the hospital the patient first noticed protrusion of the right eye, which gradually increased. Along with the increasing exophthalmos she noticed gradually failing vision. Her memory had become less acute, and occasionally she suffered from sharp, shooting pains in the left side of the thorax and the left arm. Loud noises caused buzzing sounds in the ears and dizziness. Six months before admission there developed a subjective sensation of weakness in both legs, which was not confirmed on examination at that time. For a few months she had been having generalized headaches.

Examination.—There was a large bony swelling on the right side of the skull extending from the right ear to the right orbit and from the zygoma to the upper edge of the temporal bone. There was definite right exophthalmos. The right pupil was larger than the left and was fixed to light and in accommodation. The right external rectus muscle was weak, and the right eye showed poor rotation upward and downward. Both disks presented a postpapillitic type of secondary atrophy. The margins were distinct, and the vasculature of the disks was poor. Vision in the left eye was 6/15; in the right, 6/60. The visual field of the left eye was normal; that of the right eye showed a slight concentric contraction. All deep tendon reflexes were exaggerated but equal. Roentgenographic studies revealed that the pituitary fossa measured 11 by 10 mm. There was a large, dense bony mass involving the bones of the right side of the skull

in the frontal and temporal regions. The lesion involved the roof of the orbit, the bones forming the sphenoid fissure and the base. The right olfactory groove and sphenoid ridge were thickened. There was no encroachment on the optic canals.

Operation.—In August 1933 Dr. F. C. Grant made a large bone flap in the right frontotemporoparietal region. Practically all the area of hyperostosis was removed. The dura had to be sacrificed, because it was densely adherent to that portion of the bone. When what was once the sphenoid ridge was exposed a moderate-sized meningeal fibroblastoma was seen. It extended into both the anterior and the middle fossa, but the greater part was in the anterior fossa. Most of the tumor was removed, with the exception of the part nearest the pituitary fossa. A greater part of the thickened bone was rongeured away.

Pathologic Examination.—The pathologic examination was made by Dr. B. J. Alpers. The tumor was a typical fibroblastoma arising from the meninges. The thickened bone was unusual in that the outer portion was hard and dense. The inner surface was softer, much less dense and brownish red. Examination of bits of the soft tissue removed from the bone showed strands of tumor tissue, among which were larger bands of connective tissue and numerous blood vessels.

Comment.—The neurologic findings in this case were not characteristic of a tumor of the lesser sphenoid wing. The decreased visual acuity of the right eye, with concentric contraction of the visual field, slight weakness of the right ocular muscles, postpapillitic atrophy of both optic nerves and exophthalmos were not impressive, since most of the signs could be explained by the extensive hyperostosis. The recent progressive history of failing vision in the right eye, loss of memory and contralateral subjective disturbances of sensation suggested the presence of an expanding intracranial lesion.

The tumor straddled what was at one time the lesser wing of the sphenoid bone, extending chiefly into the anterior fossa. By virtue of the anterior position of the tumor, the anterior cerebral artery may have been compressed and in that manner may have caused the loss of memory. Pressure of the tumor on the optic nerve caused the concentric contraction of the right visual field. On the other hand, the hyperostosis of the roof of the orbit was responsible for the exophthalmos and the disturbance of the ocular movements.

The interest in this case centered about the extensive hyperostosis which accompanied the tumor. This process involved the temporal and frontal bones, the orbital roof and the bones forming the sphenoid fissure and the base of the skull on the right side. Hyperostosis of the base of the skull is an uncommon finding. Spiller [4] has called attention to its occurrence in bones of the vault and has expressed the belief that it is associated with trauma. He described the case observed by Winkel-

4. Spiller, W. G.: Cranial Hyperostosis Associated with Underlying Meningeal Fibroblastoma, Arch. Neurol. & Psychiat. **21**:637 (March) 1929.

man,[5] in which there was hyperostosis of the orbital roof in connection with a large frontal fibroblastoma. That was the first reported case in which the process was confined to the bones of the base of the skull. Since then Bucy and Kredel [6] have described a case of suprasellar fibroblastoma and hyperostosis of the sella turcica. Stender [7] has reported three cases of tumor of the sphenoid ridge in which there was hyperostosis of the bone underlying the tumor. The extent of the process was not as great as in the present case. The difference between the reported cases and this case is that the hyperostosis was present for a great many years in this case before any evidence of an intracranial pathologic condition was manifest. Just what the relationship between the hyperostosis and the tumor was in this case is difficult to say.

Another example of hyperostosis of the bones underlying the tumor was seen in case 3. The process involved the sphenoid ridge and the bones in the immediate vicinity and was not as marked as in case 5. The extent of the pathologic condition was similar to that in the cases described by Stender. The occurrence of hyperostosis in tumor of the lesser sphenoid wing, as demonstrated by these two cases, emphasizes its association with tumor of the base of the skull.

The following analysis and discussion are based on the five cases which have been described here as well as on the four cases previously reported. The accompanying tables have been constructed combining the signs and symptoms in all nine cases.

ANALYSIS OF SIGNS AND SYMPTOMS

These five additional case reports not only confirm the syndrome of meningeal fibroblastoma arising from the lesser wing of the sphenoid bone by emphasizing the signs previously described but elaborate the findings, in that several additional symptoms and signs have been brought out, namely, sensory disturbances in the distribution of the trigeminal nerve, uncinate fits, exophthalmos and erosion of the lesser wing of the sphenoid bone. Furthermore, the analysis of the signs in the nine cases has given a better conception of the relative frequency of their occurrence.

History.—The history in these cases does not follow a definite chronological order. The explanation for that is obvious when the loca-

5. Winkelman, N. W.: Hyperostosis and Tumor Infiltration of Base of Skull Associated with Meningeal Fibroblastoma, Arch. Neurol. & Psychiat. **23:** (March) 1930.

6. Bucy, P. C., and Kredel, F. E.: Meningioma of the Tuberculum Sellae with Hyperostosis, Am. J. Path. **10:**805, 1934.

7. Stender, A.: Ueber das Meningiom des Keilbeinrückens, Ztschr. f. d. ges. Neurol. u. Psychiat. **147:**244, 1933.

tion of the tumors is studied. All the tumors took their origin from the dura overlying the lesser wing of the sphenoid ridge, but the manner in which they grew was not uniform. The growth extended either into the anterior fossa or into the middle fossa or into both. In two instances it extended across the midline for a short distance. On the other hand, the area of attachment varied from a very small base to one of the *en plaque* type. For that reason the structures in close relationship to the sphenoid ridge may escape pressure from the tumor, but there are certain features of the history which are worthy of emphasis.

TABLE 1.—*Summary of the Histories* *

Case	1	2	3	4	5	6	7	8	9
Site of lesion.....	Left	Left	Right	Left	Right	Right	Left	Right	Right
Duration of symptoms	3 yr.	5 yr.	1 yr.	9 yr.	1 yr.	6 mo.	1 yr.	4 yr.	5 yr.
First symptom.....	Pain in left arm	Loss of vision in left eye	Protrusion of right eye	Loss of vision in left eye	Protrusion of right eye	Failing and double vision	Failing vision	Failing vision in right eye	Failing vision in left eye
Progressive failure of vision	Left	Left	Right	Left	Right	..	Bilateral	Right	Bilateral
Unilateral protrusion of eye	Left	Left	Right	..	Right	..	..	..	..
Paresthesia in trigeminal distribution	Divisions 1 and 2	+	±	..	..	..	..	..	..
Headache.....	..	+	..	..	+	+	+	..	..
Pituitary symptoms	..	..	..	..	..	+	+	+	..
Motor symptoms....	..	..	..	..	+	..	+	..	..
Sensory symptoms..	+	..	..	..	+	..	..	..	..
Nausea and vomiting	..	..	..	..	..	..	..	..	+
Impaired memory...	..	..	..	.	+	..	..	..	..
Uncinate fits.....	..	+	..	..	..	..	..	..	..

* Cases 6 to 9 were reported in a previous article.[1]

In eight of the nine cases the clinical course extended for from six months to five years. Four of these patients had symptoms for only one year. The duration of the history, therefore, is usually short.

Failing vision, which was either unilateral or bilateral, was the most frequent initial complaint (table 1). In five cases it was recorded as the initial symptom, and in the remaining four it developed during the course of the disease. At the time of admission to the hospital one patient gave a true history of hemianopia; and another, who complained of failing vision in both eyes, had homonymous hemianopia. Unilateral loss of vision was described in the other seven cases, even though a number of the patients showed defects in the visual field of the opposite eye.

There were two instances in which unilateral exophthalmos was the initial symptom, and in two others it was recorded as a subsequent development.

Headaches were a prominent part of the history in four cases. Two of the patients showed that symptom early in the clinical course, whereas it occurred late in the other two.

Except for one case, in which paresthesia and subjective sensory disturbances in the contralateral arm were early complaints, other symptoms came late in the history. These included dysfunction of the pituitary body, motor and sensory disturbances and impairment of the memory. The uncinate fits which were noted in one case occurred before there were subjective visual disturbances.

The important facts which are revealed are that the history is usually short and that unilateral loss of vision in the majority of instances precedes other evidences of an intracranial growth, although exophthalmos and headaches may be the initial complaint. As the history

TABLE 2.—*Summary of Neurologic Findings*

Case	Atrophy of Optic Nerve	Hemianopia	Concentric Contraction of Fields	Papilledema	Exophthalmos	Hyposmia	Involvement of: Third Nerve	Sixth Nerve	Fifth Nerve	Pituitary Gland	Frontal Lobe	Motor Fibers	Sensory Fibers
1.....	+	+	..	..	+	..	+	..	+	..	..	..	+
2.....	+	+	..	..	+	..	+	+	+	..	..	..	..
3.....	+	..	+	..	+	..	..	..	..	..	..	..	..
4.....	+	+	..	..	..	..	..	..	..	..	..	..	..
5.....	+	..	+	..	+	..	..	..	..	..	+	..	+
6.....	..	+	..	..	..	..	+	..	..	..	..	..	..
7.....	+	+	..	..	..	..	..	..	..	+	..	+	..
8.....	+	+	..	+	..	+	..	..	..	+	..	..	..
9.....	+	+	..	..	..	+	..	..	..	..	..	..	..

unfolds, symptoms develop which indicate involvement of the third and fifth cranial nerves, the pituitary gland, the frontal lobe and occasionally the uncus.

Neurologic Findings.—Eye: The most striking and significant features of tumors of the lesser wing of the sphenoid bone are the ocular signs (table 2). Eight of the nine patients showed atrophy of one or both optic disks. Of these eight, five showed bilateral and three unilateral atrophy. One of the latter had papilledema of the opposite disk, representing the Kennedy syndrome. Otherwise, papilledema was not observed. The atrophy was primary in six and secondary in the remaining two patients, who had large tumors. Only one patient in this group failed to show changes in the eyegrounds, but the visual fields disclosed homonymous hemianopia. Primary atrophy of the optic nerve, therefore, is invariably present in these cases, although secondary atrophy may occur when the tumor is large.

Some defect was present in the visual field in every case (fig. 2), either homonymous hemianopia, complete or quadrantic, or a concentric contraction on the side of the tumor. There were three examples of homonymous hemianopia. Four patients were blind on the side of the lesion, with a complete or quadrantic temporal defect in the opposite field. In two there was concentric contraction of the field on the affected side. In those cases the defects occurred on the contralateral side and showed no distinguishing features which differentiated them from other

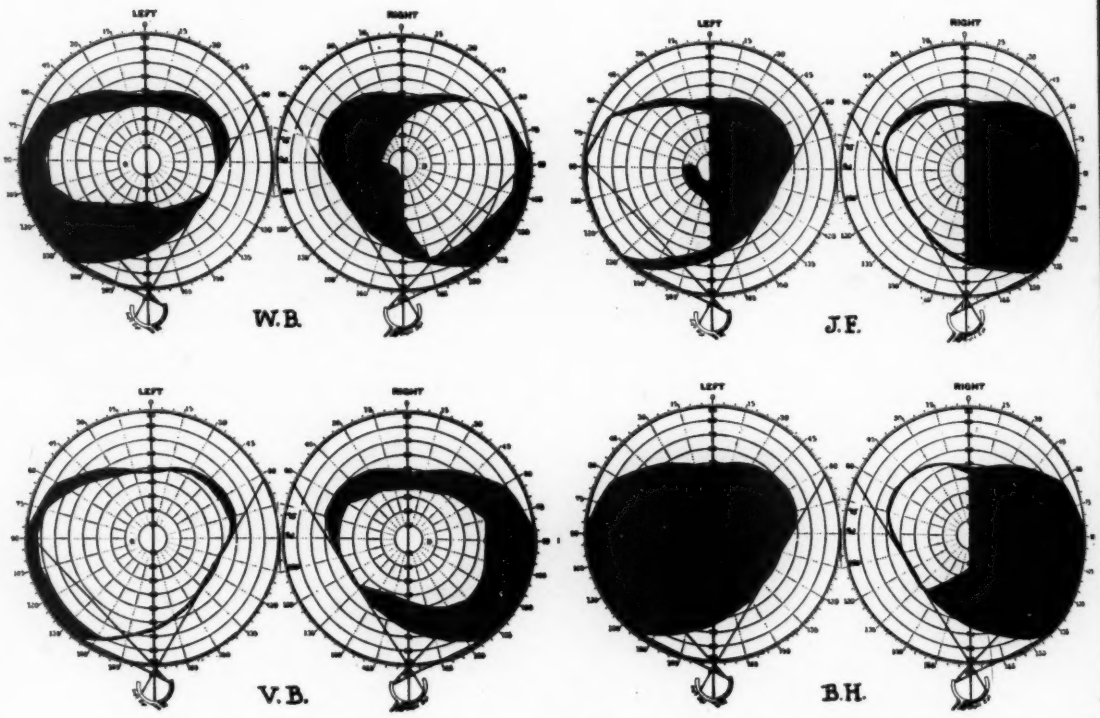

Fig. 2.—A selected group of visual fields, showing the different types. A Ferree-Rand perimeter was used, with a 1 degree white test object. W. B. (Feb. 26, 1931) had had symptoms for six months; J. F. (Oct. 7, 1930), for one year; V. B. (July 28, 1933), for one year, and B. H. (July 17, 1934), for nine years.

parasellar lesions. They were due to pressure on the optic nerve and chiasm or on the optic tract.

Exophthalmos, formerly regarded as an infrequent characteristic of the syndrome, occurred in four of the nine cases. It was always unilateral and on the side of the lesion. In two cases the explanation for this sign was hyperostosis of the orbital wall. In the remaining two no gross pathologic condition was present, and the cause was

considered to be pressure on the cavernous sinus which interfered with venous drainage from the orbit.

Oculomotor Nerve: Paralysis of the oculomotor nerve was observed in three patients and was caused by a tumor which had a paramedian position and compressed the nerve. This sign appeared after visual disturbances had been noted by the patient.

Abducens Nerve: The abducens nerve was paralyzed in one case. In another case there was weakness of the external rectus muscle, but little significance could be placed on that symptom, because of extensive hyperostosis of the walls of the orbit. Paralysis of the nerve indicates not only a paramedian tumor but one that extends rather far back in the middle fossa.

Trochlear Nerve: The trochlear, or fourth cranial, nerve was not involved in any of the nine cases.

Trigeminal Nerve: In two patients there was subjective and objective disturbance of sensation in the region supplied by the first and second divisions of the fifth nerve. One patient had pain in the ophthalmic distribution; the other had paresthesia and pain in the ophthalmic and maxillary distribution and an impaired corneal reflex. Those symptoms were explained as due to pressure on the nerve caused by a tumor which extended into the middle fossa. In a previous paper [1] this sign was not considered, but it is included now as part of the syndrome.

Olfactory Nerve: Unilateral impairment of the sense of smell was recorded in two cases. In each case the tumor had projected forward into the anterior fossa and had compressed the olfactory nerve.

Sensory System: Objective sensory disturbances were not observed in any of the nine patients; however, two had subjective sensory complaints. One patient suffered from contralateral aching and sharp, shooting pains in the arm, and the other, in the chest and arm. The final explanation for this was compression of the middle cerebral artery, with increased cortical irritability.

Motor System: In only one patient were there signs of motor weakness. This was manifested by paresis of the face and arm, with increased reflexes. In another patient there was subjective weakness in the lower part of both legs. Following operation the sensation of subjective weakness disappeared. The cause for it was not clear. The paresis of the face and arm was explained by pressure of the tumor against the middle cerebral artery, with partial occlusion, which resulted in cerebral anoxemia and death of cells. The location of the tumor prevents the corticospinal pathways from receiving pressure.

Other Signs: Disturbances of the pituitary gland were manifested in two patients by loss of libido, questionable menstrual disturbances and a gain in weight and in another by loss of libido, polyuria, a gain in

weight and cutaneous changes. In one case there was impairment of the memory, due to pressure on the anterior cerebral vessels which disturbed the function of the frontal lobe. Uncinate fits occurred in one patient. The attacks were characterized by the odor of old smoke and burning rags and were due to pressure on the uncus. Unilateral disturbance of taste was present in one patient. No explanation for it can be given.

Roentgenographic Findings.—Little can be added to the changes noted in the previous paper. Not all the cases presented alterations of the sella turcica. The changes observed (table 3) are not pathognomonic

TABLE 3.—*Summary of Roentgenographic Findings*

Case	Measurement of Sella Turcica, Mm.	Dorsum	Anterior Clinoid Processes	Posterior Clinoid Processes	Floor	Other Findings	Spinal Fluid Pressure, Mm. of Water
1	9×11	Negative	Negative	Negative	Negative	Erosion of left sphenoid wing	...
2	12×11	Negative	Negative	Negative	Negative	Calcified mass posterior to pituitary on left	...
3	15×12	Negative	Negative	Erosion	Erosion	Erosion of right sphenoid wing and right optic foramen	110
4	13 very shallow	Negative	Negative	Negative	Negative	.....	140
5	11×10	Negative	Thickened	Negative	Negative	Large osteoma of frontal and temporal bone and sphenoid ridge	...
6	12×11	Rarefied	Negative	Negative	Negative	Calcification along clivus	140
7	12×6	Slight atrophy	Some pointing	Negative	Negative	.....	160
8	.....	Complete erosion	Negative	Complete erosion	Thin and depressed	.....	320
9	12×11	Negative	Negative	Negative	Thin curved shadow about pit	.....	...

of these tumors, but when they are present they aid in establishing the diagnosis. They are in agreement with the observations of Kornblum [8] for the parasellar group of tumors as represented by those in the middle fossa.

The sella turcica was "top normal" in size in six of the nine cases. The term "top normal" is used, according to the work of Kornblum, to signify that the pituitary fossa measures approximately "from 8 to 10 mm. in depth and 10 to 12 mm. in the anteroposterior diameter." The dorsum was completely eroded in one case and rarefied and slightly

8. Kornblum, Karl: Deformation of the Sella Turcica in Tumors of the Middle Cranial Fossa, Am. J. Roentgenol. **31**:23, 1934.

eroded in two others. In one case there was complete erosion of the posterior clinoid processes.

In two cases there was a unilateral erosion of the sella turcica. The appearance in the roentgenogram was that of a double floor. In one case this was associated with erosion of the dorsum, the posterior clinoid processes and the floor. In the other case there was erosion of the floor and of the posterior clinoid processes. This sign is an extremely valuable part of the syndrome, but it occurs in cases of tumor of the middle fossa as well. Thus it cannot be considered as characteristic of a tumor of the lesser sphenoid wing alone.

Fig. 3 (case 3).—Roentgenogram showing enlargement of the sella turcica (15 by 12 mm.) and a double line for the floor, which is indicative of unilateral erosion.

A feature that was not observed previously but was present in two of the cases reported here was erosion of the lesser wing of the sphenoid bone. This sign is likewise helpful when the clinical picture suggests the presence of a lesion in that region but is not limited to these tumors.

Erosion of the optic foramen on the side of the lesion was reported in one case. This sign, as stated previously, might be of considerable importance. Not all the patients were examined for this change, so it cannot be said how frequently it occurs. Pancoast [9] has studied its

9. Pancoast, H. K.: The Roentgen Diagnostic Significance of Erosion of the Optic Canals in the Study of Intracranial Tumors, Ann. Surg. **101**:246, 1935.

Fig. 4 (case 3).—Roentgenogram showing erosion of the right sphenoid ridge, indicated by the arrows. This film was made postoperatively, the defect having been noted at operation. This view offers the best visualization of this structure.

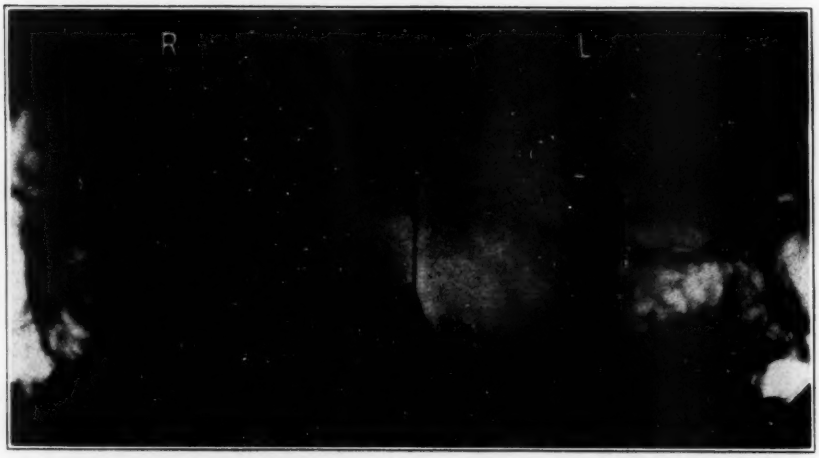

Fig. 5 (case 3).—Roentgenogram showing the optic foramina, indicated by the arrows. Note the erosion of the right optic foramen.

localizing value and has observed erosion of the optic foramen in other types of tumors and in tumors not arising from the sphenoid ridge. He expressed doubt about its significance with regard to lateralization of the tumor, since in one of the cases there was erosion of the optic canal on the side opposite the lesion. He was of the opinion that if the clinical localization is in doubt, especially as to laterality, this sign should be disregarded and either ventriculography or encephalography should be performed.

Other evidences of localization supplied by the roentgenograms were calcification in the tumor, demonstrated in one patient, and hyperostosis of the sphenoid ridge and adjacent bones, observed in two others. Hyperostosis of the bones of the skull is rare and for that reason commands special attention. In one case it was definitely associated with the tumor. In the second case the hyperostosis occurred many years before there was any indication of an intracranial pathologic condition and was first observed in the temporal bone. Obviously, a definite connection between the tumor and the pathologic state of the bone cannot be established in this case, yet tumor cells were recovered from the bone. These two examples, especially the first, emphasize the occurrence of hyperostosis in bones of the base and, together with Stender's cases, establish its relationship with meningeal fibroblastoma of the sphenoid ridge.

My experience with encephalographic study has been limited to that of one patient who had a large tumor. The pictures suggested a suprasellar lesion, and lateralization was based on the clinical picture. In early cases in which the roentgenograms do not reveal any abnormality localizing encephalographic studies may be extremely valuable. The work of Elsberg and Dyke [10] has shown that there is unilateral deformity of the cisterna interpeduncularis on the side of the tumor. This observation is a distinct advancement in the early recognition of a tumor of the lesser sphenoid wing, but great caution should be exercised in the interpretation of the cisternal air markings.

COMMENT AND SUMMARY

The syndrome of meningeal fibroblastoma of the lesser wing of the sphenoid bone is a definite entity. The clinical picture usually begins with unilateral loss of vision, and that is a prominent and progressive symptom throughout the course of the disease. In some instances unilateral exophthalmos may precede the visual disturbances. Later, symp-

10. Elsberg, C. A., and Dyke, C. G.: Meningiomas Attached to the Mesial Part of the Sphenoid Ridge, with Syndrome of Unilateral Optic Atrophy, Defect in Visual Field of Same Eye and Changes in Sella Turcica and in Shape of Interpeduncular Cistern After Encephalography, Arch. Ophth. **12**:644 (Nov.) 1934.

toms develop that are referable to the structures in close relationship to the lesser wing of the sphenoid bone.

The neurologic findings in the early stage may be limited to primary atrophy of the optic nerve, which may be a homolateral, quadrantic or homonymous contralateral defect in the visual field and in some instances homolateral exophthalmos. Roentgenograms at this stage usually do not reveal any abnormality but may show a "top normal" sella turcica.

In the more advanced stage there is primary atrophy of both optic nerves, rarely secondary atrophy, and the visual fields show contralateral homonymous hemianopia or blindness on the homolateral side and a temporal defect in the opposite field. Further neurologic findings depend on the direction in which the tumor grows. In case of paramedian extension there is involvement of the oculomotor nerve; evidences of dysfunction of the pituitary gland, such as a gain in weight, a loss of libido, menstrual disturbances and cutaneous changes, and infrequently paralysis of the abducens nerve. Extension of the tumor into the middle fossa produces signs referable to the first and second divisions of the trigeminal nerve, and in some instances uncinate fits occur. Anterior extensions may compress the olfactory nerve, causing unilateral hyposmia and rarely symptoms referable to the frontal lobe. A large tumor may give rise to contralateral subjective sensory or motor signs.

The roentgenograms may demonstrate one or more of the following changes: a "top normal" sella turcica, unilateral erosion of the sella turcica, erosion of the lesser wing of the sphenoid bone, erosion of the optic foramen on the side of the lesion and, in rare instances, hyperostosis of the sphenoid ridge and adjacent bones.

DIFFERENTIAL DIAGNOSIS

Among the lesions which develop close to the lesser wing of the sphenoid bone there are four which might be confused with the tumor under consideration, i. e., a suprasellar tumor, an intrasellar growth with parasellar extension, an aneurysm of the internal carotid artery or of its branches and an intra-orbital growth. In cases of the first type of lesion the visual fields usually show a bitemporal defect without other evidences of involvement of the cranial nerves such as are seen when there is a growth in the lesser sphenoid wing. On the basis of these facts a differential diagnosis can be made; but, in spite of this, auxiliary aids may be necessary.

An intrasellar tumor with parasellar extension invariably presents a definite picture of dysfunction of the pituitary gland, and the roentgenogram will disclose changes in the pituitary fossa which are more in keeping with those of a primary tumor of the pituitary gland.

An aneurysm of the internal carotid artery or of its branches in this location offers a more serious problem. The clinical history of one or more attacks of hemorrhage, a more focal type of erosion of the pituitary fossa or of the lesser wing of the sphenoid ridge and calcification of the internal carotid artery, if present, usually suffice to establish the diagnosis of an aneurysm.

Recently there came under the observation of Dr. F. C. Grant a patient who presented much the same clinical signs and symptoms described in the syndrome of tumor of the sphenoid ridge. The partial ptosis, unilateral exophthalmos, primary atrophy of the optic nerve with complete blindness, all on the right side, roentgenographic evidence of a "top normal" sella turcica and changes in the lesser wing of the sphenoid bone brought up the possibility of the presence of a tumor of the lesser sphenoid wing. The only finding that was not in keeping with this diagnosis was weakness of all the muscles of the affected eye. The diagnosis rested between that of a tumor of the sphenoid ridge and an intra-orbital growth. The case is reported in detail.

Right intra-orbital tumor. Symptoms confined to right eye for two years. Exophthalmos of right eye with marked ptosis. Visual acuity in right eye 3/60; left eye 6/12. Complete right temporal hemichromatopsia; left field irregularly contracted. Slight limitation of ocular movements, especially outward rotation. Right corneal anesthesia and anesthesia of first division of right fifth nerve. Pituitary fossa, 14 mm. by 10 mm. Lateral erosion of dorsum sellae and involvement of sphenoid bone.

History.—O. H., a man aged 50, was admitted to the service of Dr. F. C. Grant in the Graduate Hospital, having been referred by Dr. Dryer, of Allentown, Pa., and Dr. C. A. Patten, of Philadelphia. He complained of failing vision in the right eye. He was well until the fall of 1933, when he noticed a burning sensation over the right palpebral fissure. In the summer of 1934 the vision in the right eye became blurred. Coincidentally the right side of the forehead felt numb. By November 1934 the right upper lid began to droop. During that time the vision in the right eye gradually decreased until the man was barely able to distinguish between light and dark.

Examination.—Neurologic findings were limited to the right eye. There were definite exophthalmos and almost complete ptosis of the right eye. The right pupil was smaller than the left and did not react to light and in accommodation. Visual acuity in the right eye was 1/60; that in the left eye, 6/9, corrected. There was a complete temporal cut to red in the right visual field and irregular concentric contraction of the left. The right optic disk presented complete secondary atrophy; the left was normal. Ocular rotations were limited in all directions but especially outward. The corneal reflex on the right was diminished, and there was complete anesthesia of the first division of the right fifth nerve. The pituitary fossa measured 14 mm. by 10 mm. There appeared to be lateral erosion of the dorsum sella and involvement of the sphenoid bone. In addition, there was a defect in the bony outline of the anterior cranial fossa on the right side, just in front of the anterior clinoid process. Examination of the optic foramen revealed no abnormality.

Operation.—On Jan. 12, 1935, Dr. F. C. Grant performed a transfrontal craniotomy on the right side. Exploration of the right sphenoid ridge revealed a thin and fragmented inner end, but no tumor was seen. The chiasm and the right optic nerve appeared normal. The roof of the orbit was removed, and a tumor measuring 2.5 by 2 cm. was disclosed in the posterior part of the orbit, lateral to and above the optic nerve just before it passed through the foramen. One of the ocular muscles disappeared into the tumor. The growth was completely removed.

Comment.—In retrospect, the diagnosis in this case can be made clinically. The misleading factor was the indefinite and confusing roentgenographic findings, which favored a diagnosis of tumor of the sphenoid ridge. The changes in the roentgenograms were confirmed at operation and were readily explained by the presence of the retro-orbital growth. The deciding point in the differential diagnosis between a tumor of the sphenoid ridge and an intra-orbital tumor was that in a case of intra-orbital tumor there is relatively equal paresis of all ocular muscles and no definite defects are seen in the visual field. In this case there was a secondary type of atrophy of the optic nerve. However, in some cases the diagnosis may be extremely difficult, and encephalographic studies are then necessary.

SURGICAL COMMENT

In cases of fibroblastoma of the lesser wing of the sphenoid bone the surgical problem is not difficult if the tumor is small. As stated in previous notes and emphasized by Elsberg and Dyke,[10] the surgical approach should be from the side of the lesion. This permits adequate exposure and easy accessibility of the growth. Each case is necessarily a problem of its own; but a lateral approach is particularly advantageous, since it allows exposure of the portion which is in the middle fossa and the dural attachment is removed with greater ease. The one difficulty which arises when the tumor is large is in connection with the adjacent internal carotid artery and its branches. These vessels are so obscured that great care must be exercised in removing the most mesial portion. The dural attachment must be removed in order to prevent a recurrence of the growth. In this clinic when technical difficulties prevent complete removal of the dura it is customary to coagulate the dura thoroughly with the electrosurgical unit. In carrying out this procedure, great care should be taken not to injure the structures passing through the superior orbital fissure.

THE ROYAL LONDON OPHTHALMIC HOSPITAL

A VISIT TO THE KING GEORGE V EXTENSION

BERNARD SAMUELS, M.D.
NEW YORK

It is said in England that hospitals are being rebuilt, enlarged and founded on a scale reminiscent of the sixteenth century almshouses. However, such shelters are no longer for "twelve poor men and twelve poor women headed by a governor or governess"; the wealthy and well-to-do are now clamoring for the advantages of a hospital which are not to be had at a nursing home. In keeping with the spirit of the times the present year at Moorfields has been marked by the completion and dedication on May 16 of a large building which His Majesty the King graciously consented to have named the King George V Extension.

As a preliminary to the construction of the extension under survey in this paper, a scheme of alterations in the original structure was carried out five years ago, by which a separate ward unit for private patients, including an operating theater and a sterilizing room, were provided, together with additional rooms for nurses and the domestic staff. The completion of so great an undertaking was made possible by gifts, mostly expressed in small sums, representing true charity, not the least among the contributors being patients in the clinic, who gave according to their means.

An idea of the size and equipment of the King George V Extension may be gained from the following list of units contained within its walls:

1. The outpatient department, with its auxiliary services, e. g., refraction department, spectacle room, dispensary, social service department and registration and inquiry offices.
2. Theater for minor operations and casualty department.
3. Pathologic department.
4. Bacteriologic department.
5. Physical therapy department.
6. Orthoptic department.
7. Medical school, embodying lecture theaters, the library and the museum.
8. Additional rooms for private patients.

9. Cubicle ward beds.

10. Additional accommodations for the nursing and the domestic staff.

The hospital in its present form is a vast and impressive pile, albeit two distinct schools of architecture stand forth in its construction. The old portion, completed in 1899, conforms to the French renaissance style, with its elegancies, ornamentations and pediments. The new portion boldly exemplifies the modern school, with its main facade of buff-colored terra cotta and severe straight lines, making no effort to conceal its utilitarian purpose, in fact, emphasizing it. The Extension comprises five stories, as does the old part. It is constructed of steel frame, with double floors of reenforced concrete, between which are the appliances for lighting, steam-heating and drainage. On the north side each story from the third downward is set back in order to insure

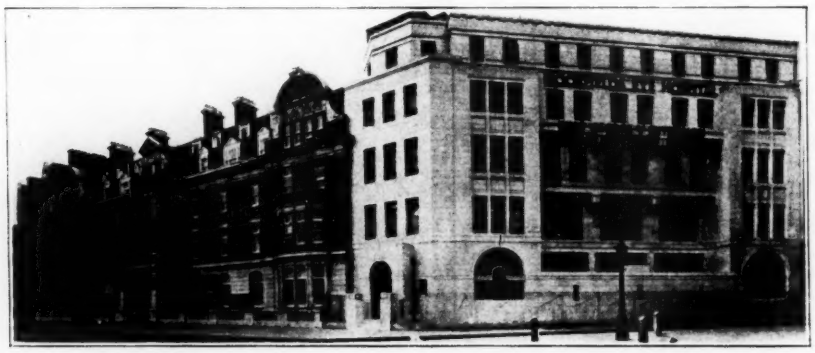

Fig. 1.—The Royal London Ophthalmic Hospital.

the necessary light for the ground floor. With a view to obtaining a still greater measure of light the walls of the open areas are faced with glazed white brick. A note of beauty and dignity, symbolic of the high calling of the edifice, appears over the porch of the entrance in the form of a sculpture in half-relief of Christ giving sight to blind Bartimaeus.

THE GROUND FLOOR

The main problem involved in the planning of the ground floor was to furnish space for the handling and treatment of a maximum number of 750 outpatients daily, the daily average for 1934 being 548. Entirely separate room is provided for five surgeon's clinics, each self-contained, with its own cubicles for ophthalmoscopic, slit-lamp and perimetric examination. In each surgeon's consulting room there are two desks, supplied with an electric lamp and a control panel to enable the surgeon to manipulate and illuminate a test chart and an astigmatic fan. Patients waiting without are summoned by lights, new patients responding to

one color and old ones to another. In retrospect, in comparison with these conveniences and amenities, one recalls the erstwhile vast consulting room at Moorfields, in which four distinct groups of surgeons stood, surrounded by assistants and students, in the midst of an expanse of benches filled with on-looking patients.

In the arrangement of the outpatient department the greatest effort has been made to insure that the general flow of patients shall be in one direction (fig. 3). There are two patients' waiting halls, one for the new patients and one for the old ones. Immediately on arrival they are segregated. The new patients are conducted to the issuing office, where they receive their medical notes and a patient's card. Next,

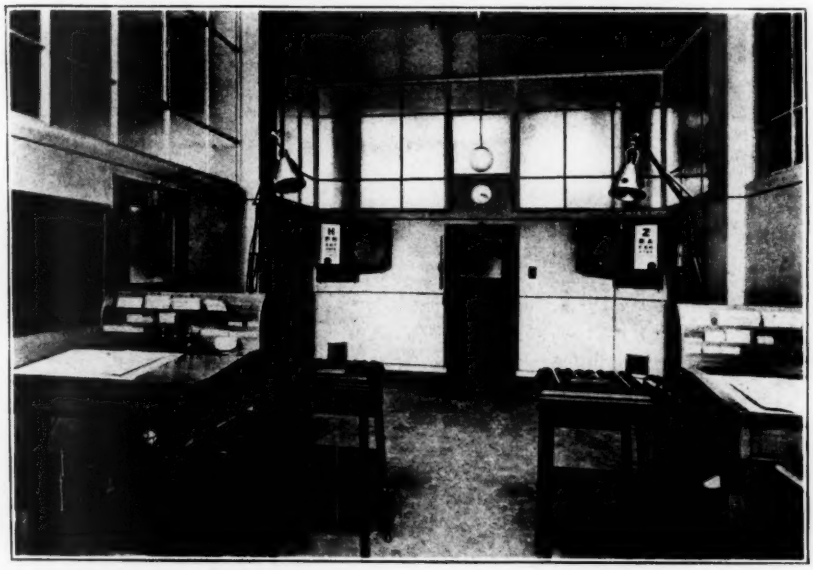

Fig. 2.—A surgeon's clinic.

they go to the registration office, where full clerical details are noted, and then to the inquiry office, from which they are directed to the various departments (fig. 4). The old patients proceed directly to the medical notes store, where on presentation of their card they are handed their medical record. The decision as to which clinic or department a patient is to enter is made by a physician, called the "sifting" medical officer. To assist patients to find their way, colored signs corresponding to the color of the cards issued have been installed at convenient points. Similarly, patients referred by a surgeon in one of the clinics to any of the special departments, namely, orthoptic, physical therapy, bacteriologic or pathologic, are given cards so colored as to correspond to signs indicating the route to be taken. This arrangement has been found to facilitate the flow of patients to the various branches and departments.

Fig. 3.—The main waiting hall in the outpatient department.

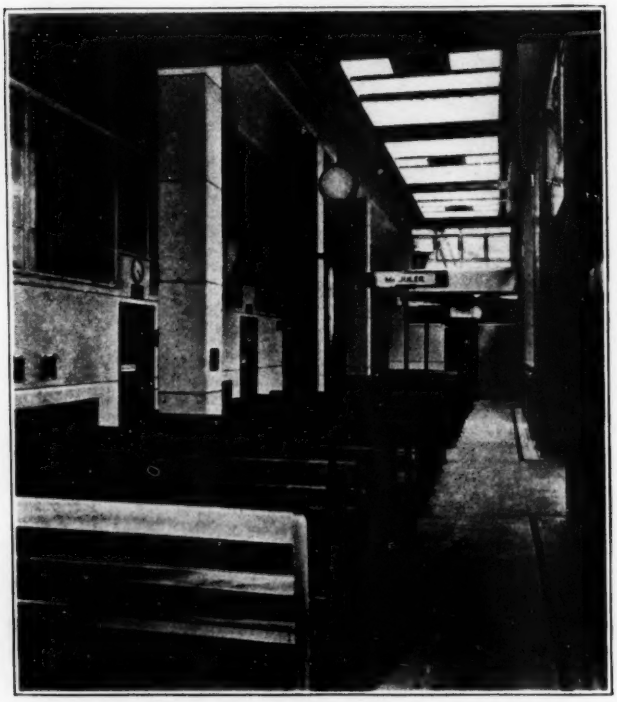

Fig. 4.—The waiting space outside a surgeon's clinic.

Patients, both old and new, on leaving a surgeon's clinic, the refraction department or the department for minor operations, proceed to the dispensary, the spectacle room or the social service department.

A system that may commend itself has been adopted by which new patients are interviewed by the lady almoner after they have been examined and treated by a surgeon, and not before, on the principle that the social service aspect of a case can be better served after some knowledge of the physical condition of the patient has been ascertained. It is left to the lady almoner to ascertain the financial status of the patients and to assess them accordingly.

Fig. 5.—The refraction department.

The Medical Records.—The records of outpatients are filed in specially constructed cabinets provided with ball-bearing extension arms. Speed of access to the records is of prime importance, since in one morning four or five hundred records have to be extracted from the files. The medical record of every outpatient is kept in the filing store for seven years, so that nearly 350,000 records are on file at one time. A hand basin, supplied with hot and cold water and a special antiseptic, is provided to protect the filing clerks when handling records of patients suffering from trachoma.

The Refraction Department.—The space allotted to tests for refraction is divided into seventeen cubicles, so darkened as to enable the surgeon to examine the fundi. Without moving the patient the examiner

is able to make a refraction test, using a test type 6 meters away on the wall of the opposite cubicle. Some 20,000 prescriptions for spectacles are written a year. Two outpatient medical officers serve in each of the four clinics as refractionists. They are remunerated for their services. The hospital neither makes nor sells spectacles, but it arranges for an optician to be in attendance daily.

Ventilation.—The system of ventilating the vast outpatient department has received special attention. An air-conditioning plant, installed in the basement, consists of a large centripetal fan which draws air from outside. The air, after having been thoroughly cleansed, is heated

Fig. 6.—The waiting space outside the refraction department.

or cooled as required. During its passage the air comes in contact with a finely divided water spray, which serves to cleanse it and, when desired, to humidify it. The freshly tempered air is then forced through vertical flues placed at numerous points. During hot dry weather a decided cooling effect is added to the atmosphere. The whole plant is designed on such ample lines that no matter how crowded the outpatient department may become the ventilation is adequate to insure the comfort of both the patients and the staff. Particular thought has been given to the refraction cubicles. Here the air can be completely changed every ten minutes. Provision is made for the exhaust of vitiated air as a whole by way of the great dome overhead in the waiting hall.

THE FIRST FLOOR

On the first floor are the orthoptic and physical therapy departments. As is known far and wide, at Moorfields of recent years much study and research have been devoted to the subject of squint. This department is commodious and is fully equipped with expensive instruments. It is in charge of a medical officer, trained in this particular field, to whom the surgeons in the various clinics refer patients for diagnosis and training, each patient being returned to the parent clinic from time to time for final disposition. About 20 patients can be handled at one time. This department was at first placed on a research basis for

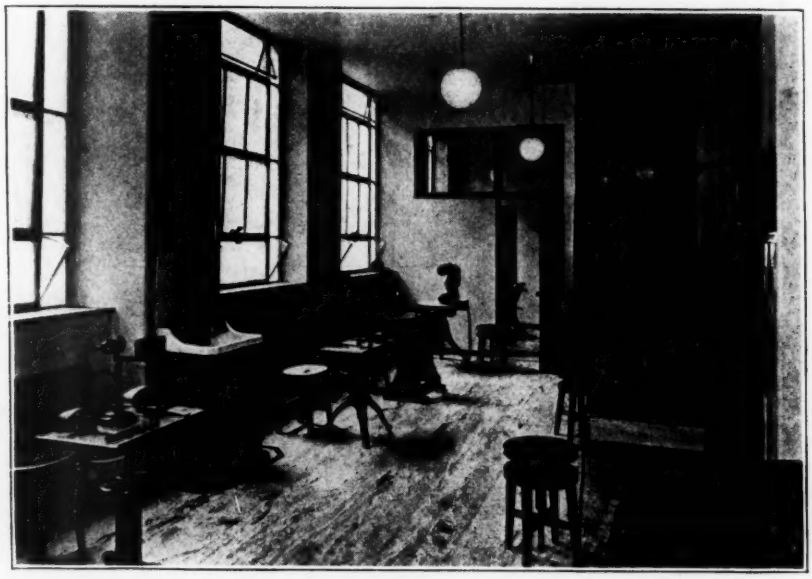

Fig. 7.—The orthoptic department.

two years, as a result of which it has now been incorporated as a permanent department of the hospital.

The Physical Therapy Department.—The physical therapy department possesses a carbon arc lamp for general radiation therapy, a mercury vapor lamp for both general and local application and an apparatus to provide diathermy, ionization and electrolysis. Ultraviolet rays are administered not only in a concentrated localized form to the eye but also to the entire body as a general tonic.

The Library and Museum.—On the first floor are also the library and the museum. The library is of such abundant space that it can accommodate periodicals on ophthalmology for twenty years to come. Some of the books are of great historical interest. It is a handsome,

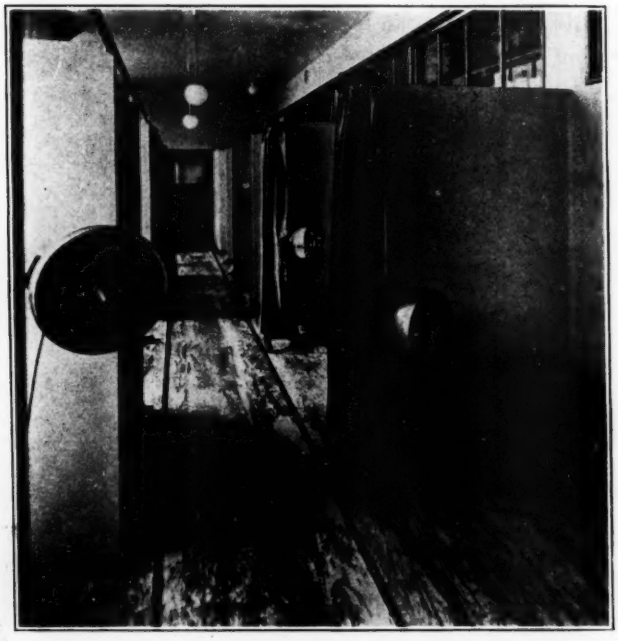

Fig. 8.—The ultraviolet ray department.

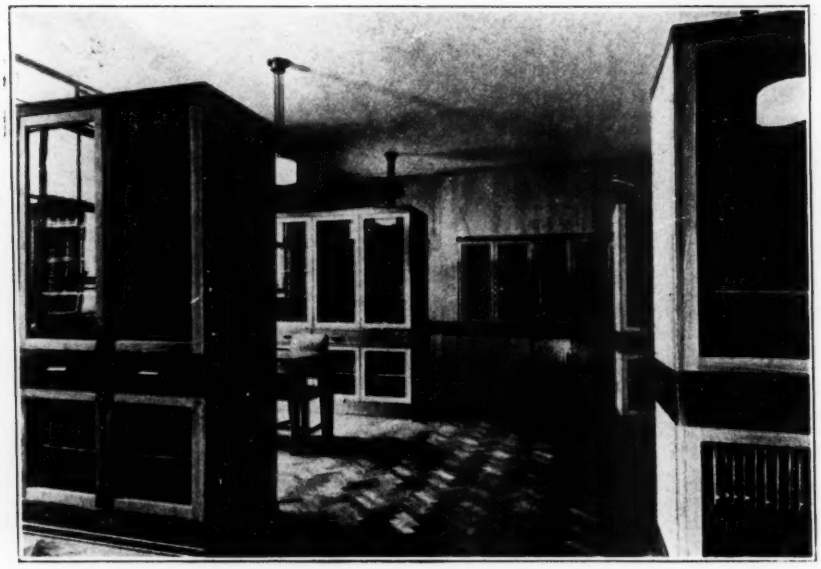

Fig. 9.—The library.

airy room, with bookcases of oak and walls of oak veneer. The floor is of teak blocks. The library is cataloged. It is maintained and kept up to date by means of a library fund.

The museum houses a great number of gross and microscopic specimens and drawings assembled over a long period of years. It is unique in the possession of a comprehensive collection of specimens on comparative anatomy, which is constantly being enriched. The library and museum are under the direction of the pathologist and curator, who serves for five years and is paid a salary.

THE SECOND FLOOR

The Pathologic and Bacteriologic Departments.—The pathologic and bacteriologic departments are on the second floor. The large laboratories of these departments are unusually well equipped for conducting graduate teaching. Every eye or piece of tissue removed in the operating rooms is sent to the pathologic department as a routine procedure. About 300 globes are examined in the course of a year, a number of them being sent in from other parts of the country and from abroad.

The bacteriologic department covers a wide field. It reports on smears and cultures from the conjunctiva in practically all cases before operation. Corresponding to 600 operations for cataract performed in the hospital in 1934, 1,200 cultures were made, the practice being to make a culture twice before an operation for removal of a cataract. The records of the bacteriologic department show that since the adoption in the operating room of dry sterilization of instruments, the maintenance of dry hands and the use of dry dishes the loss of eyes from sepsis is very rare, being about 1 in 1,000. Not only are blood tests and other ordinary tests made and serum and vaccine treatments given but special clinics are held here by the head of the department for the treatment of syphilitic and gonorrheal diseases that manifest themselves in the organs of vision.

The Main Lecture Hall.—The main lecture hall is on the second floor, providing accommodation for eighty students. Included in the equipment is an epidiascope for the demonstration of specimens stereoscopically and in color. The room is finely proportioned, with walls paneled in silvered oak veneer.

THE THIRD FLOOR

On the third floor the house surgeons and nursing staff are provided with dining-rooms, which cannot help but elicit praise for their graceful lines and color schemes. The remainder of the floor is given over to fourteen bedrooms for nurses and to a large common room for them. Every bedroom is provided with a built-in wardrobe, a lavatory basin, with hot and cold water, and a bedside light. The furniture is of

oak. Facilities for ironing are at the disposal of the nurses, the electric irons having a timing-switch and pilot-light to lessen the risk of fire. The walls of the nurses' common room are of Australian walnut, and the floor is of oak blocks. A loud speaker is built in the paneled walls.

THE FOURTH FLOOR

The fourth floor provides twenty-three nurses' bedrooms and a sitting-room for private patients. The walls of the latter are of Australian walnut, and the floor is covered with "korkoid," which is much used in other parts of the building.

COMMENT

All doors throughout the extension are of oak, flush-faced, and have a lining of granulated cork to insure that noise is reduced to a minimum. Esthetically, the extension breathes an air of comfort and cheerfulness.

The underlying policy at Moorfields would seem to center in a disposition to make the hospital a self-centered unit. A dining-room and a sitting-room are provided for students who attend the hospital. There is a buffet for the outpatients, at which they may obtain meals and light refreshments, and it is interesting to note that a substantial profit accrues to the hospital from this source.

The installation of a chapel is contemplated. Finally, the hospital has under its management an ophthalmic convalescent home for children in the country outside London.

The Royal London Ophthalmic Hospital is to be congratulated on carrying to completion a scheme of such a comprehensive nature as the King George V Extension, which it is said ranks as the largest and finest clinic of its kind in the world. This is the more impressive at a time when it has been argued that the old independent, special hospitals should be merged in the great medical centers. The strength of Moorfields probably lies in the variety of its interests and activities, for it is not only a hospital but a school of ophthalmology. The University of London and the Conjoint Board for England recognize it as a place of instruction in ophthalmology, and all members of the honorary staff are classed as teachers in the university. It comprises perhaps the largest teaching staff of ophthalmology in any country. During the year 1932-1933, 400 lectures were delivered to 120 students.

The King George V Extension stands as a happy omen that Moorfields not only will be able to maintain but may even enhance its proud reputation; for here ripened the knowledge of Saunders, Travers, Lawrence, Dalrymple, Hutchinson, Bowman, Nettleship, Collins, Parsons and others, who by devotion to duty and by the printed word have made of it the "storied metropolis" of ophthalmology in the English-speaking world.

THE PUPILLARY REACTIONS IN COMBINED LESIONS OF THE POSTERIOR COMMISSURE AND OF THE PUPILLODILATOR TRACTS

A CONTRIBUTION TO THE PATHOGENESIS OF THE ARGYLL ROBERTSON PUPIL

NORMAN P. SCALA, M.D.
Instructor of Ophthalmology, Georgetown University
WASHINGTON, D. C.
AND
ERNEST A. SPIEGEL, M.D.
Professor of Experimental and Applied Neurology, Temple University
PHILADELPHIA

Attempts to localize the anatomic lesion in cases of the Argyll Robertson pupil were recently concentrated on the posterior commissure, a system of fibers that cross the midline dorsal to the sylvian aqueduct in the most cranial part of the mesencephalon. Particularly the anatomic studies of Lenz [1] and Papez and Freeman [2] and the experiments of Sachs [3] and of Ranson and Magoun [4] show that afferent fibers of the pupilloconstrictor light reflex arc enter the posterior commissure and have here at least a partial decussation. Thus the theory of Behr [5] seemed very promising; this author explains the double-sided Argyll Robertson pupil by a lesion of the pupillomotor fibers at the place of decussation in the midbrain and the unilateral Argyll Robertson pupil by a lesion between the decussation in the midbrain and the sphincter nucleus, although Karplus and Kreidl [6] had elicited pupillary contraction

From the Department of Experimental Neurology, D. J. McCarthy Foundation, Temple University School of Medicine, Philadelphia.

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1. Lenz, J.: Klin. Monatsbl. f. Augenh. **79**:875, 1927; Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:140, 1927.

2. Papez, J. W., and Freeman, G. D.: J. Comp. Neurol. **51**:409, 1930.

3. Sachs, E.: Brain **32**:95, 1909.

4. Ranson, S. W., and Magoun, H. W.: Central Path of Pupilloconstrictor Reflex in Response to Light, Arch. Neurol. & Psychiat. **30**:1193 (Dec.) 1933; Proc. Soc. Exper. Biol. & Med. **31**:183, 1933; Central Path of Light Reflex: Study of Effect of Lesions, Arch. Ophth. **13**:791 (May) 1935.

5. Behr, C.: (*a*) Die Lehre von den Pupillenbewegungen, Berlin, Julius Springer, 1924; (*b*) Zentralbl. f. d. ges. Ophth. **14**:465, 1925.

6. Karplus, I. P., and Kreidl, A.: Arch. f. d. ges. Physiol. **149**:115, 1913.

by stimulation of the brachium of the superior colliculus even after incision into the posterior commissure. The theory of Behr is "accepted by a majority of students and writers on this subject," according to the review of the Argyll Robertson pupil given by McAndrews.[7] Sven Ingvar,[8] assuming a marginal destruction of the pupillomotor pathways in the surface layers of the diencephalon, explains the unilateral Argyll Robertson pupil in agreement with Behr's views. Recently Merritt and Moore[9] advanced an interesting modification of Behr's theory, trying to outline a lesion that could explain all the phenomena of the Argyll Robertson pupil. Wilson[10] already supposed that the miosis in Argyll Robertson pupils may be due to a lesion of the pupillodilator tracts in the midbrain. The theory of Merritt and Moore follows a similar direction. These investigators referred to the experiments of Beattie, Brow and Long,[11] who studied the course of the pupillodilator fibers originating in the hypothalamus. After lesions of this region these authors found by the degeneration method descending fibers on the ventral and lateral aspect of the gray matter around the aqueduct. The fibers enter the midbrain by passing ventrally to the fibers of the posterior commissure. Part of the fibers end in the superior colliculus, and part arch ventrally and pass caudally to the lower parts of the central nervous system. Merritt and Moore, therefore, assumed a unilateral or bilateral lesion of the posterior commissure between its decussation and the sphincter nucleus encroaching also on the neighboring pupillodilator fibers that descend from the hypothalamus. "There would be a loss of the light reflex owing to involvement of the light reflex fibers, and a myotic pupil which would not react to painful stimuli and which would dilate imperfectly to atropine solutions owing to involvement of the descending pupillodilator fibers." These authors believe that "this explanation of the production of the Argyll Robertson pupil rests on a firm anatomic foundation and explains the phenomena better than any other heretofore offered." Indeed this theory merits special interest, as it endeavors to explain not only the loss of the light reflex, but also the symptoms associated with the Argyll Robertson pupil, such as miosis, the failure

7. McAndrews, L. F.: Argyll Robertson Pupil, Arch. Ophth. **10**:520 (Oct.) 1933.

8. Ingvar, Sven: Bull. Johns Hopkins Hosp. **43**:315, 1928.

9. Merritt, H. H., and Moore, M.: Argyll Robertson Pupil: Anatomic-Physiologic Explanation of Phenomenon, with Survey of Its Occurrence in Neurosyphilis, Arch. Neurol & Psychiat. **30**:357 (Aug.) 1933.

10. Wilson, S. A. K.: Modern Problems in Neurology, London, Edward Arnold and Co., 1928.

11. Beattie, J.; Brow, G. R., and Long, C. N. H.: Proc. Roy. Soc., London, s.B **106**:253, 1930; A. Research Nerv. & Ment. Dis., Proc. **9**:249, 1930.

to dilate in response to painful stimuli and the imperfect dilatation after the instillation of atropine.

The question arises how lesions within the area outlined by Merritt and Moore affect the various reactions of the pupil. In a former study one of us (Spiegel [12]) observed that the direct as well as the consensual light reflex of the pupil can still be elicited in cats after median or paramedian severance of the posterior commissure. Thus the results of these experiments were not in agreement with Behr's theory. The experiments, however, were performed before Merritt and Moore's modification of this theory was published, and the latter theory, particularly the eventual rôle of lesions of the centrifugal fibers from the hypothalamic centers, could not yet be taken into consideration. It seemed desirable, therefore, to carry on a further series of experiments in which the severance of the posterior commissure was combined with the lesion of the pupillodilator tracts that descend from the hypothalamus, in order to study the light reflex as well as the dilator reactions of the pupils after lesions in this region. Unilateral lesions have the advantage as compared with bilateral injuries, as they permit the comparison of the pupil on the side on which the operation has been performed with that of the opposite pupil. By such a comparison small disturbances may be detected that escape observation after bilateral lesions.

METHOD

The experiments were performed on ten cats. After the pupillary reactions were tested, the parietal and occipital bones were trephined under ether anesthesia, and the occipital lobe was exposed. By introducing small pledgets of cotton between the occipital lobe and the falx and the tentorium, respectively, the occipital lobe was gradually pushed outward. In this way the roof of the midbrain was exposed. Injury of the vena magna (Galeni) had to be avoided. The superior colliculus was punctured with a small lancet in its most cranial part, either in the midline, after carefully pushing aside the vena magna by small pledgets of cotton, or 2 or 3 mm. laterally from the midline. In order to prevent the knife being thrust farther than intended, it was enclosed in a shield, so that only the length to be introduced into the midbrain remained bare. The depth of the puncture was from 4 to 6 mm. In some experiments, after puncturing the corpora quadrigemina close to the midline, the knife was moved a few millimeters laterally so that a larger part of the midbrain was destroyed. Other modifications of the operative procedure will be described later (cats 5 and 6). After the wound was closed and the animal had recovered from the anesthesia the size of the pupils was measured. The direct and consensual pupillary reflex, the reaction on psychic excitation (e. g., strapping) and painful stimuli (faradic stimulation of the sciatic nerve) were tested; in some experiments eventually the effect of instillation of a 1 per cent solution of atropine sulfate was also studied. The extent of the lesion was examined on serial sections stained by Weil's method.

12. Spiegel, E. A.: Further Experiments on Localization of Argyll Robertson Phenomenon, Vol. jubilaire, Marinesco, 1933, p. 625.

RESULTS AND COMMENT

The experiments had to answer two questions: first, how do lesions or how does severance of the posterior commissure influence the light reflex of the pupil, and, second, is the dilatation of the pupil by psychic excitation, painful stimuli and atropine, respectively, lost or lessened after lesions to the centrifugal fibers that descend from the hypothalamus?

In regard to the first question, one has to bear in mind that the light reflex of the pupil is a reaction that is easily impaired or abolished if the central nervous system is in a poor condition owing to narcosis, asphyxia, shock and other factors and that then even lesions outside the pupillary reflex arc, e. g., in the rhombencephalon, sometimes induce a loss of this reflex. Experiments proving that the pupillary light reflex is retained after injury to certain parts of the central nervous system are therefore more conclusive in regard to the localization of the reflex arc and the pathogenesis of the Argyll Robertson pupil than lesions that abolish the light reflex. The first type of lesions permits one at least to ascertain which areas of the central nervous system are dispensable for the maintenance of the pupillary light reflex and to exclude them as possible sites of the lesions in the Argyll Robertson pupil.

In our material (see table) the light reflex could no longer be elicited in three cases (3, 7, 10). The loss of this reflex could be easily explained

TABLE 1.—*Effect of Lesions on Pupillary Reactions in Ten Cats*

	Anatomic Extent of the Lesion	Symptoms
Cat 1.	Severance of the posterior commissure on the left side; the lesion passed through the gray matter around the aqueduct and ended on the right side ventral to the ventrolateral border of the gray matter	Diameter of right pupil 4 mm. and that of left pupil 2 mm.; protrusion of left nictitating membrane; direct light reflex: right pupil contracted from 4 to 2 mm., and left pupil from 2 to 1 mm.
Cat 2.	Descending limb of the posterior commissure was severed on the left side; in the level of the mamillary bodies the lesion lay close to the left wall of the third ventricle (fig. 3A); on sections through the superior quadrigeminal bodies it extended more and more in the lateral parts of the central gray matter and encroached on the dorsal part of the reticular formation (fig 3B).	Diameter of right pupil 2.5 mm. and that of left pupil a slit; protrusion of left nictitating membrane; on stimulation of sciatic nerve: left pupil dilated from 1 to 4 mm., and right pupil from 2.5 to 6 mm.; on psychic excitation (strapping of the animal): left pupil dilated from a slit to 1 mm., and right pupil from 1 to 4 mm.; light reflex: during the dilatation on painful stimulation direct and consensual reaction demonstrable
Cat 3.	Severance of the posterior commissure; extensive lesions of the central gray matter, encroaching on both oculomotor nuclei and the roots of the right oculomotor nerve	No light reflex; no reaction on stimulation of sciatic nerve
Cat 4.	Incision into the left superior quadrigeminal body, destroying its lateral part and the descending limb of the posterior commissure; the lesion encroached on the lateral part of the gray matter around the aqueduct	Diameter of pupils: right 6 mm., left 5 mm.; right 4.5 mm., left 4 mm.; right 7 mm., left 6 mm.; right 7 mm., left 5 mm.; slight protrusion of left nictitating membrane; direct light reflex: right pupil contracted from 6 to 2 mm., and left pupil from 5 to 4 mm.;consensual reaction present on both sides; on stimulation of sciatic nerve: right pupil dilated from 7 to 9 mm., and left pupil from 6 to 8 mm.; for effect of atropine see table 2

TABLE 1.—*Effect of Lesions on Pupillary Reactions in Ten Cats—Continued*

	Anatomic Extent of the Lesion	Symptoms
Cat 5.	I. Hemorrhage in the left superior quadrigeminal body, destroying the fibers of the posterior commissure on this side in the level of the pineal gland (fig. 1)	After the first lesion: direct light reflex was contraction of left pupil from 8 to 6 mm., and of right pupil from 8 to 4 mm.; later: left pupil contracted from 9 to 6 mm., and right pupil from 7 to 3.5 mm.; maximal dilatation of both pupils on psychic excitation
	II. Incision into the floor of the fourth ventricle in the cranial part of the pons on the right side (fig. 4); the fiber systems in the central gray matter and in the dorsal part of the reticular formation were severed	After the second lesion: diameter of left pupil 6 mm. and that of right pupil 4 mm.; slight protrusion of right nictitating membrane; on stimulation of sciatic nerve: dilatation of left pupil from 6 to 8 mm. and of right pupil from 4 to 6 mm.; direct light reflex: right pupil contracted from 4 to 2 mm., and left pupil from 5 to 3.5 mm.; consensual light reflex: right pupil contracted from 6 to 3 mm., and left pupil from 7 to 5.5 mm.
Cat 6.	I. Destruction of the left ganglion habenulae; paramedian severance of the most cranial fibers of the posterior commissure only; in its posterior parts this commissure was intact, but a hemorrhage in the tectum of the superior quadrigeminal body pressed on it from dorsally	After the first lesion: diameter of right pupil 5 mm. and that of left pupil 5.5 mm.; later, diameter of right pupil 4.5 mm. and that of left pupil 6.5 mm.; direct light reflex was contraction of left pupil from 5.5 to 3 mm., and of right pupil from 5 to 2.5 mm. (slow contraction of left pupil); later, left pupil contracted from 6.5 to 4.5 mm., and right pupil from 4.5 to 3 mm.; consensual light reflex: left pupil contracted from 6 to 4.5 mm., and right pupil from 5 to 3 mm.; on psychic excitation: left pupil dilated to 8.5 mm., and right pupil to 7.5 mm.
	II. Incision into the floor of the fourth ventricle through the nucleus triangularis, which was destroyed, and through the dorsal part of the tegmentum of the pons and midbrain; the midline was not reached, and the posterior longitudinal bundle was left intact	After the second lesion: diameter of right pupil 5 mm., and that of left pupil, 7 mm.; direct light reflex: left pupil contracted from 7 to 5 mm., and right pupil from 5 to 3 mm.; on stimulation of sciatic nerve: left pupil dilated from 7 to 9 mm., and right pupil from 5 to 8.5 mm.
Cat 7.	Two incisions severing the posterior commissure on the right side, extending through the central gray matter on the right side of the aqueduct and reaching the oculomotor nuclei	Light reflex absent; both pupils contracted spontaneously from 7 to 2 mm.; on psychic excitation dilatation to 6 mm. was observed; instillation of atropine dilated the right pupil to 5 mm. and the left to 8 mm.
Cat 8.	Large hemorrhage in the tectum of the left superior quadrigeminal body, destroying the descending limb of the posterior commissure; the lateral part of the gray matter around the aqueduct was destroyed on sections through the nucleus ruber	Diameter of left pupil 6 mm. and that of right pupil 4.5 mm.; on psychic excitation: both pupils dilated to 9 mm.; direct light reflex: left pupil contracted from 6 to 4 mm., and right pupil from 5 to 3 mm.; consensual light reflex: left pupil contracted from 5 to 4 mm., and right pupil from 4 to 3 mm.
Cat 9.	Severance of the posterior commissure on the right side, ending in a hemorrhage in the dorsolateral parts of the gray matter around the caudal part of the third ventricle (fig. 2)	Diameter of left pupil 7 mm. and that of right pupil 8 mm.; very slight light reflex; two days later, diameter of both pupils 8 mm.; direct light reflex: left pupil contracted from 8 to 6 mm., and right pupil from 8 to 7 mm.; consensual reaction present in both eyes
Cat 10.	Large hemorrhage severed the commissure on the left side, extended through the periventricular gray matter on the left, destroyed also the roots of the third nerve on the left and pressed on the third nucleus on the right	Diameter of left pupil 3 mm. and that of right pupil 4 mm.; later, diameter of left pupil 5 mm. and that of right pupil 1 mm.; on stimulation of sciatic nerve: left pupil dilated from 5 to 9 mm., and right pupil from 1 to 6 mm.; similar dilatation on psychic excitation (strapping of the animal); light reflex absent

in these cases, as the anatomic examination showed extensive injuries reaching to the oculomotor nuclei and nerves, respectively. In the remaining seven experiments the light reflex could still be observed after the lesions to the posterior commissure. These lesions were paramedian in cats 1, 5, 6 and 9, affecting the posterior commissure close to its decussation in the zone of transition from the midbrain to the diencephalon.

While in cases 1, 5 and 9 the commissure was completely severed (figs. 1 and 2), in case 6 only the most cranial part was cut, and more caudally the hemorrhage in the roof of the superior colliculus compressed the commissure from the dorsal side. In cases 2, 4 and 8 the lesion was located in the descending limb of the commissure more or less close to the oculomotor nucleus. According to Behr's theory, one should expect a loss of the pupillary light reflex on the side of the lesion of the posterior commissure. However, the direct as well as the consensual reaction to light was still retained on both sides. The amplitude of the pupillary contraction was usually larger (cases 1, 4, 5 and 9) on the

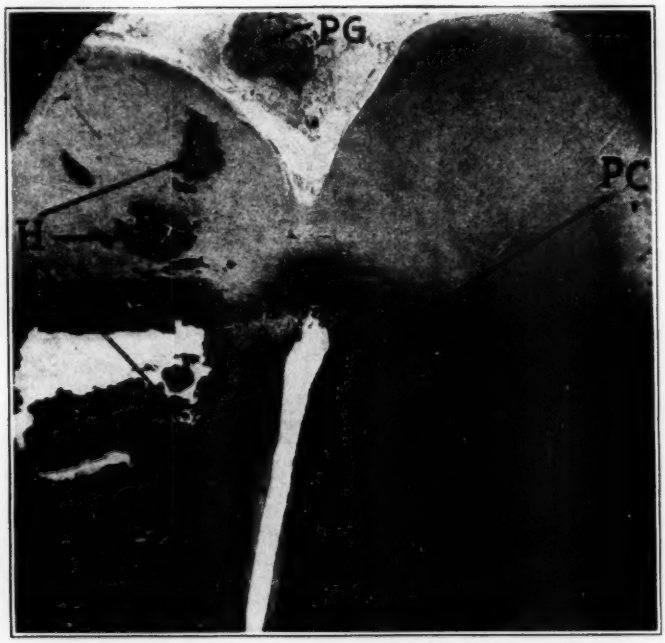

Fig. 1 (cat 5).—Section showing hemorrhage (H) in the midbrain. *PC* indicates the posterior commissure and *PG* the pineal gland.

normal side than on the side on which the operation was performed. In cat 6 the pupil on the injured side contracted also somewhat slower than the normal pupil. In cat 2 bilateral miosis developed after the lesion, apparently owing to an irritative effect of the lesion on the sphincter nucleus, so that the light reflex could at first hardly be observed. It could, however, be shown if the pupils were dilated by painful stimuli (stimulation of the sciatic nerve) or psychic excitation. Then it could be seen that the illumination induced a direct as well as a consensual pupillary contraction.

If one surveys these experiments, they seem in agreement with the theory that the posterior commissure carries pupillary constrictor

impulses from the optic tract toward the sphincter nucleus, as the light reflex was diminished on the side of the lesion of the posterior commissure.[13] The fact that the pupillary reflex to light is not abolished after unilateral severance of the posterior commissure on the homolateral pupil might be explained in two ways. The intact limb of the posterior commissure might carry optical impulses to both sphincter nuclei,[14] or the pupillary constrictor pathways might be located only partly within the posterior commissure, while another part might be situated laterally to the posterior commissure. As to the first probability, Ranson and Magoun [4] observed that the stimulation in the region through which the fibers of the posterior commissure ran ventrad and caudad around the central gray matter elicits not only contraction of the homolateral pupil

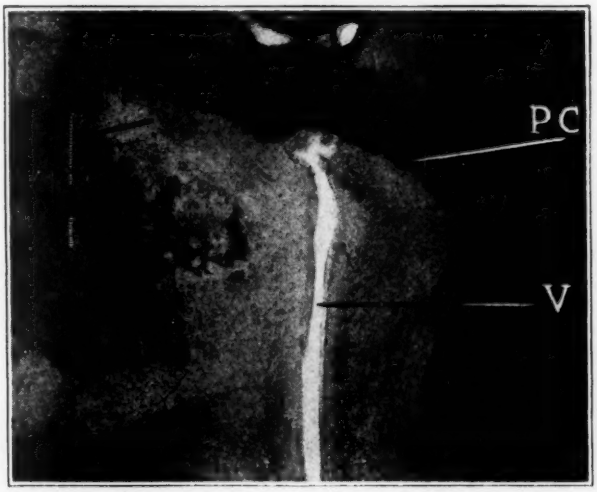

Fig. 2 (cat 9).—Section showing severance (*L*) of the posterior commissure (*PC*) on the right side. *V* indicates the third ventricle.

13. After unilateral severance of the optic tract (Karplus and Kreidl [6]) or of the afferent path of the light reflex in the pretectal region (Ranson and Magoun [4]) of cats the light reaction of the opposite eye was more severely impaired than that of the homolateral eye, apparently because the greatest part of the pupilloconstrictor fibers cross in the chiasma. We found after severance of the posterior commissure between its decussation and the sphincter nucleus a weaker reaction of the homolateral pupil to the light stimulus. This indicates that the major part of the pupilloconstrictor impulses returns by crossing in the posterior commissure to the original side.

14. The term sphincter center or sphincter nucleus is here used for the sake of brevity. Yet it is probable that the oculomotor fibers innervating the sphincter muscle originate not only in the most cranial part of the Edinger-Westphal nucleus (the nucleus medianus anterior) but also in small cells within the lateral oculomotor nucleus (for details see Spiegel, E.: Zentren des autonomen Nervensystems, Berlin, Julius Springer, 1928).

but also a weak contralateral constriction. They are inclined to assume that a second smaller crossing takes place ventral to the aqueduct.[15]

In regard to the second possibility, i. e., the existence of a part of the pupilloconstrictor pathways outside of the posterior commissure, in some former experiments of one of us (Spiegel [16]) extensive injuries of the gray matter around the aqueduct were produced in order to study the eventual part of the central gray matter in the pathogenesis of the Argyll Robertson pupil. It was found that such injuries of the central gray matter did not abolish the light reflex. A reexamination of these lesions showed that they encroached in some cases (rabbits 19 and 22 of the paper of Spiegel and Nagasaka) on the posterior commissure on both sides and that the light reflex of the pupil could still be elicited. Furthermore the stimulation experiments of Ranson and Magoun [4] seem to indicate that constriction of the pupil can be obtained not only from the posterior commissure but also from a zone adjoining it laterally. If these effects were due only to a spread of the current on the fibers of the posterior commissure, constriction of the pupil would result also from stimulation of an equally large zone on the inner side of the commissure. This was not observed. All these facts indicate that a part of the pupilloconstrictor pathways takes its way laterally to the posterior commissure. Yet a further study of the question seems necessary.

In regard to the Argyll Robertson pupil, a lesion of the posterior commissure close to its decussation or of the descending limb is not sufficient to produce an abolition of the light reflex in the ipsilateral eye. If one wants to explain the loss of the light reflex by a lesion of the centripetal part of the reflex arc, one has to assume either an extensive unilateral or bilateral lesion, including besides the fibers of the posterior commissure the extracommissural pupilloconstrictor pathways, and the second ventral partial decussation of the reflex arc, respectively, or a lesion close to the cells of the sphincter nucleus where all the optic pathways converging toward this nucleus can be easily affected by one single lesion. The first assumption of an extensive lesion seems improbable, if one considers that examination with the usual histologic methods in the overwhelming majority of cases of Argyll Robertson phenomenon

15. They leave it undecided as to whether this is a crossing of secondary optic fibers or of oculomotor fibers. As to the human brain, it seems to us improbable that a crossing of fibers arising in the sphincter nucleus takes place to an extent of any practical importance. One has to bear in mind that cases of unilateral Argyll Robertson pupil were observed and that this symptom can be produced only by a lesion between the last decussation in the light reflex arc and the sphincter muscle. If this last decussation or semidecussation is peripheralward from the sphincter nuclei, the lesion causing the unilateral Argyll Robertson pupil should be in the fibers of the oculomotor nerve or in the neuron of the ciliary ganglion. This, however, is rather improbable, as is pointed out later.

16. Spiegel, E. A., and Nagasaka, G.: Arch. f. d. ges. Physiol. **215**:120, 1926.

fails to show lesions of this structure. The second assumption of a small lesion around the cells of the sphincter nucleus could explain without difficulty not only the double-sided but also the unilateral appearance of the Argyll Robertson pupil, as such a pericellular lesion could shut out impulses from the homolateral and the contralateral secondary optical pathways.

One has to bear in mind, however, that the Argyll Robertson pupil is usually associated with miosis, while the interruption of the optic pathways not only abolishes the pupillary light reflex but causes also a dilatation of the pupil. In fact, after a lesion of the posterior commissure the homolateral pupil was in our experiments slightly wider than the contralateral pupil, if these lesions were not complicated by destruction of the pupillodilator tracts (cat 6 after the first lesion and cat 9). This leads to the second question as to whether some of the symptoms of the Argyll Robertson pupil, particularly miosis, the abolishment of the dilator reaction on painful stimuli and psychic excitation and the imperfect dilatation in response to atropine can be produced by a lesion of the centrifugal tracts that arise in the hypothalamus. It is well known (F. H. Lewy and Beattie, Brow and Long [11]) that lesions of the vegetative hypothalamic centers or of their centrifugal pathways are followed by miosis. Here, however, the question arises how the pupil is changed if the severance of the hypothalamic pupillodilator tracts is combined with that of the pupilloconstrictor pathways in the posterior commissure.

If one studies the influence of such lesions on the width of the pupil, one must of course exclude cases in which the lesions encroached on the oculomotor nuclei or their fibers (cats 3, 7 and 10) or in which the pupilloconstrictor and the dilator pathways were severed on different sides (cat 5). The other cases may be divided into two groups. In the one the lesion had a sufficient extent [17] to destroy the descending

17. Ingram, Ranson and Hannett (Am. J. Physiol. **98**:687, 1931) obtained pupillary dilatation by electrical stimulation from practically anywhere in the reticular formation of the tegmentum. Yet it cannot be determined from their observations how far these effects were due to stimulation of ascending or of descending tracts, as they observed dilatation of the pupil also in stimulation of the medial lemniscus and the spinothalamic tracts. Beattie, Brow and Long damaged in their experiments not only the periventricular gray matter but also the nucleus tuberomamillaris (personal communication to the authors), the region from which Ranson and Magoun (Respiratory and Pupillary Reactions Induced by Electrical Stimulation of Hypothalamus, Arch. Neurol. & Psychiat. **29**:1179 [June] 1933) obtained dilatation of the pupil. The objection of these authors seems, therefore, not to invalidate Beattie's work. If one assumes that the pupillodilator tracts descending from the hypothalamus occupy a larger area than outlined by Beattie, Merritt and Moore's theory of the Argyll Robertson pupil could not be taken into consideration, because this theory is based on Beattie's work and supposes a small lesion adjacent to the posterior commissure.

pupillodilator tract, as described by Beattie, Brow and Long;[11] in the other a part of the pathways remained intact.

First, in regard to the last group (cats 6, 8 and 9), in cat 6 the lesion of the floor of the fourth ventricle and of the tegmentum pontis did not reach far enough medialward to destroy all the fibers described by Beattie, Brow and Long. In cat 8 the lesion affected the lateral part of the central gray matter around the aqueduct and the tectum of the superior quadrigeminal body only in levels caudal to the posterior commissure. According to Beattie, Brow and Long, some of the descending hypothalamic pathways enter in this level the dorsal part of the tegmentum of the midbrain, and this part apparently escaped the lesion. In cat 9 the lesion ended closely ventral to the posterior commissure and was apparently not large enough to sever the dilator tract.

In this group of cases the pupil on the side on which the pupilloconstrictor fibers were severed was wider than that on the unaffected side. On the contrary, in that group in which the combined destruction of the pupilloconstrictor fibers of the posterior commissure and of the neighboring dilator fibers was accomplished without lesion of the oculomotor nuclei or fibers (cats 1, 2 and 4) the injury to the two antagonistic systems of the pupil resulted in a slight miosis (the pupil on the side of which the operation was performed was from 1 to 2 mm. smaller than that on the other side). Thus if the lesion of the pupillodilator hypothalamic systems supervenes on that of the afferent fibers of the pupillary light reflex, the dilatation of the pupil resulting from the latter lesion may be prevented, and even a slight miosis may result. A high degree of miosis is usually not observed after such an operation or at the most only a short time after the operation, owing to an irritative effect on the sphincter nucleus.

The experiments have furthermore to ascertain whether destruction of the pupillodilator pathways that descend from the hypothalamus abolishes the dilatation of the corresponding pupil in response to emotional or painful stimuli, as Merritt and Moore assumed. Painful and emotional stimuli were still able to produce dilatation of both pupils, despite the unilateral destruction of the pupillodilator fibers that descend from the hypothalamus. This dilatation had, for instance, the same extent on both sides in cat 4 (2 mm.), although the inequality of the pupils (the pupil on the side on which the operation had been performed was 1 mm. smaller) was still present when both pupils were dilated owing to the painful stimulation. In cat 2 (figs. 3*A* and *B*) the pupil dilated 0.5 mm. less on the side on which the operation had been performed than on the normal side when the sciatic nerve was stimulated; but the amplitude of the dilatation was still 3 mm. on the side on which the operation had been performed. On psychic stimulation (strapping of the animal) the affected pupil dilated from a slit to 1 mm., the normal pupil from 1 to 4 mm.

In order to produce as complete a destruction as possible of the fibers that descend from the hypothalamus without injuring by such an extensive lesion the oculomotor nuclei or nerves, in some experiments the floor of the fourth ventricle was exposed after opening the atlanto-occipital membrane. An incision was made on one side into the cranial part of the floor of the fourth ventricle to destroy the pupillodilator tracts that, according to Beattie, Brow and Long, are located at the level of the inferior colliculus on the floor of the fourth ventricle, where they become confused with the ascending vestibular fibers and with the dorsal reticular formation. In the pons they become more and more concentrated toward the median plane. In cat 5 the lesion was located in the cranial part of the pons on the right side, severing here the tracts

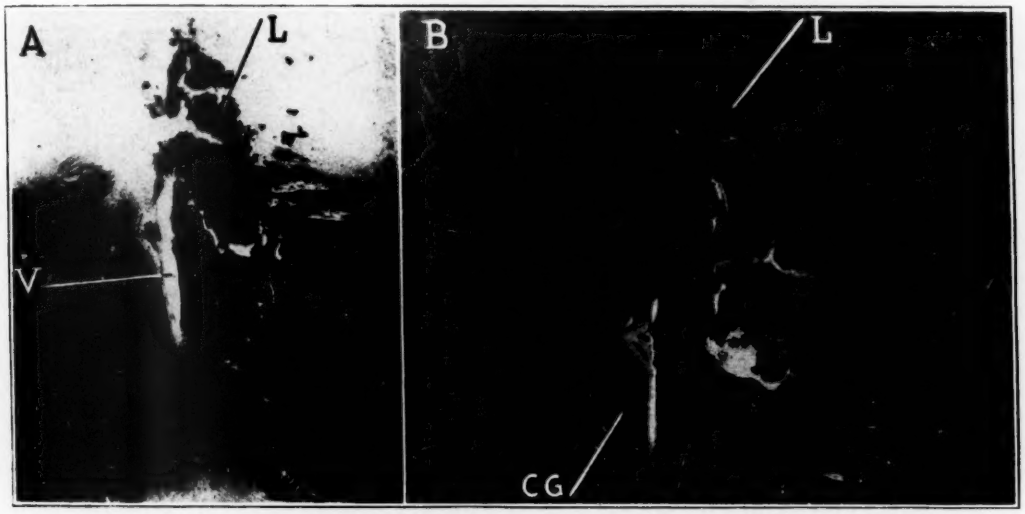

Fig. 3 (cat 2).—*A*, section through level of the mamillary bodies; the lesion (*L*) lies close to the left wall of the third ventricle (*V*). *B*, section through the superior quadrigeminal bodies (*S*); the lesion (*L*) lies in lateral parts of the central gray matter (*CG*) and the dorsal part of the tegmentum.

in the central gray matter and in the dorsal part of the reticular formation (fig. 4). After this lesion also the dilator reaction of the pupil was well preserved on either side, although the pupil on the side of the lesion remained smaller than the opposite pupil, also at the maximum of the dilatation. On stimulation of the sciatic nerve the right pupil dilated from 4 to 6 mm., the left pupil from 6 to 8 mm. The severance of the descending vegetative pathways from the hypothalamic centers was shown by the protrusion of the nictitating membrane besides the results of anatomic examination and the miosis.

The result produced by unilateral severance of the pupillodilator fibers descending from the hypothalamus is only a relatively slight

impairment of the dilatation of the homolateral pupil in response to painful or emotional stimuli, but by no means a loss of this reaction, as can be observed in cases of Argyll Robertson pupil. The objections might be raised that the maintenance of the dilatation of the pupil in response to painful stimuli after unilateral lesions to the centrifugal vegetative hypothalamic tracts is at least in part due to the existence of further semidecussations of these vegetative tracts caudally from the place of the lesions (decussation in the medulla oblongata or in the cervical cord). The existence of such semidecussations is probable, as has already been shown by Karplus and Kreidl.[18] Yet if one admits

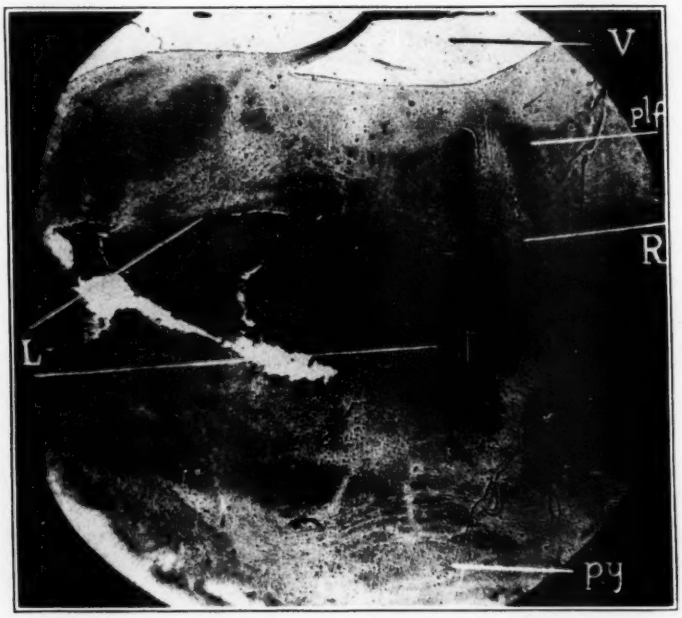

Fig. 4 (cat 5).—Section showing ventral end of the incision into the floor of the fourth ventricle in the cranial part of the pons (right side). *Plf* indicates the posterior longitudinal fasciculus; *py*, the pyramidal tract; *R*, the raphe, and *V*, the fourth ventricle.

the existence of such decussating fibers, lesions of the centrifugal vegetative tracts from the hypothalamus in the midbrain can never produce the symptoms of unilateral Argyll Robertson pupil with loss of the pain reaction of this pupil. In other words, this objection confirms only our view that the loss of pain reaction in cases of the Argyll Robertson pupil cannot sufficiently be explained by lesions in the region of the central gray matter around the aqueduct.

18. Karplus, I. P., and Kreidl, A.: Arch. f. d. ges. Physiol. **143**:109, 1911.

One has furthermore to bear in mind that the dilatation of the pupil in response to painful or emotional stimuli is performed not only by stimulation of the dilator muscle of the pupil but also by reciprocal inhibition of the antagonist, the sphincter muscle of the pupil. In former experiments carried on under the direction of one of us (Spiegel) by Gozzano [19] the severance of both cervical sympathetic nerves was combined with extirpation of the roof of the midbrain and the central gray matter around the aqueduct until close to the oculomotor nuclei. Yet stimulation of the sciatic nerve still yielded dilatation of the pupil. Some authors (Luchsinger [20] and Anderson [21]) succeeded even under certain conditions in producing dilatation of the pupil by stimulation of sensory spinal nerves after high transverse section of the cord. In these cases the sensory impulses could not reach the higher parts of the central nervous system above the spinal cord. An intraspinal reflex action of sensory impulses on the spinal center of the

TABLE 2.—*Effect of Lesions on Reaction of Pupil to Atropine in Cat 4*

Time	Right Pupil	Left Pupil
	Diameter, 7 mm.	Diameter, 5 mm.; protrusion of the left nictitating membrane
12:36	Instillation of 3 drops of a 1 per cent solution of atropine in each eye	
12:44	Diameter, 9 mm.	Diameter, 7 mm.
	Instillation of 3 drops of solution of atropine in each eye repeated	
12:55	Diameter, 9 mm. Light reflex positive	Diameter, 9 mm. Light reflex positive
12:55	Instillation of 3 drops of solution of atropine in each eye repeated	
1:02	Diameter, 9.5 mm. Light reflex absent	Diameter, 9 mm. Light reflex absent

cervical sympathetic nerve may, therefore, also play a part in the dilatation of the pupil in response to painful stimuli. Lesions near the posterior commissure affecting the centrifugal pathways that descend from the hypothalamus are therefore not sufficient to produce the loss of the dilator reaction of the pupil in response to psychic or painful stimuli.

The effect of such central lesions on the reaction of the pupil to atropine is still less distinct. The protocol for cat 4 gives an example of such an experiment (lesion of the hypothalamic tracts on the left side at the level of the posterior commissure and of this commissure).

As this protocol shows, although before the instillation of atropine and in the first minutes of the toxin's action the affected pupil was 2 mm. smaller than that on the normal side, it finally reached nearly the same extent of dilatation as did the opposite pupil. In other words, the affected pupil reacted more slowly under the influence of the atropine, but only to a somewhat smaller extent than the normal pupil. This

19. Gozzano, M.: Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:644, 1925.
20. Luchsinger: Arch. f. d. ges. Physiol. **22**:158, 1880-1881.
21. Anderson: J. Physiol. **30**:15, 1904.

may be explained by the fact that after unilateral destruction of the pupillodilator tracts in the midbrain the homolateral centrum ciliospinale still receives some tonic impulses from the opposite side, so that the homolateral pupil, although smaller than the pupil on the opposite side, does not entirely lack a tonic innervation from the cervical sympathetic.

Thus our experiments show that the combination of unilateral severance of the posterior commissure with destruction of the adjacent pupillodilator tracts may result in slight degrees of miosis but only slight impairment of the dilatation of the pupil in response to painful stimuli; the dilator effect of atropine may be retarded, but its extent is only slightly diminished, and finally nearly the same extent of dilatation as that in the opposite pupil may be reached.

Thus lesions of the sympathetic pupillodilator fibers in the midbrain may play at most an auxiliary part in the pathogenesis of the miosis and the changes of the dilator reactions of the pupil, but they cannot explain the loss of dilatation of the pupil in response to painful stimuli as observed in advanced cases of Argyll Robertson pupil. Furthermore, we have seen that a lesion of the centripetal part of the light reflex arc must be either rather extensive or close to the sphincter nuclei in order to abolish the light reflex. In view of the negative anatomic findings, an extensive lesion is improbable, while a small lesion immediately around the sphincter nuclei would not reach the pupillodilator tracts. Hence anatomically also the theory that all the symptoms of the Argyll Robertson pupil are due to combined lesions of the secondary optic pathways and of the pupillodilator tracts meets difficulties.

CONCLUDING CONSIDERATIONS ON THE PATHOGENESIS OF THE ARGYLL ROBERTSON PUPIL

The question arises where the lesion has to be sought that can explain all the symptoms of the Argyll Robertson pupil. Theories that localize the lesion outside the reflex arc of the light reflex have only historical interest. In regard to lesions within the reflex arc the points under consideration can be grouped in the following way:

1. Lesions of the centrifugal part of the light reflex arc. These are (*a*) lesions of the sphincter nucleus (Levinsohn [22]), (*b*) lesions of the oculomotor fibers (Bauer [23]) and (*c*) lesions of the ciliary ganglion (Marina [24]). An objection against these theories has already been made by Redlich,[25] who maintained that lesions of the motor neuron innervating the sphincter muscle cause mydriasis, while the Argyll Robertson pupil is in the majority of cases associated with miosis. Furthermore,

22. Levinsohn, G.: Deutsche Ztschr. f. Nervenh. **56**:300, 1917.
23. Bauer, J.: Deutsche Ztschr. f. Nervenh. **61**:144, 1918.
24. Marina, A.: Ann. di nevrol. **19**:209, 1903.
25. Redlich, E.: Wien. klin. Wchnschr. **35**:756, 1922.

histopathologic changes in the motor parts of the reflex are missed in the majority of the cases of the Argyll Robertson phenomenon.

2. Lesions of the central gray matter around the aqueduct (Monakow, Marburg,[26] Wilson [27] and Redlich [25]).

The conception that the fibers carrying optical impulses to the sphincter nucleus take their way through the central gray matter around the aqueduct and are here interrupted in cases of Argyll Robertson pupil must be abandoned, because extensive injuries to the gray matter around the aqueduct do not abolish the light reflex (Spiegel and Nagasaka [16]), and stimulation of this region does not yield constriction of the pupil (Ranson and Magoun [4]).

3. Lesions of the centripetal part of the reflex arc (marginal degeneration of the optic pathways [Sven Ingvar [8]]), lesions of the secondary optic pathways in the midbrain at the decussation or between it and the sphincter nucleus (Behr [5a] and Merritt and Moore [9]) and selective degeneration of the optic reflex collaterals around the sphincter nucleus (Bumke [28]). Injuries to the optic tract, to the superior quadrigeminal branch or to the continuing fibers before their decussation in the midbrain need no consideration, as they cause a different syndrome, namely, hemianopic loss of the pupil reaction. Bumke's assumption of a selective degeneration of optic reflex collaterals leads, as has already been pointed out by Bauer,[23] to the improbable assumption that in cases of unilateral Argyll Robertson pupil all the optic reflex collaterals from both optic tracts should degenerate around one sphincter nucleus and the corresponding collaterals should remain intact around the opposite oculomotor nucleus. Theories that explain the Argyll Robertson pupil by lesions of the posterior commissure are refuted by the fact that severance of this commissure *does not* abolish the pupillary light reflex. Magoun and Ranson [4] found that lesions of the pretectal region impair or abolish the light reflex, but they emphasize that such lesions produce no Argyll Robertson pupil. Purely unilateral loss of the light reflex could not be produced by such lesions. The pupils even dilated widely after a transitory stage of miosis.

The miosis and the loss of the dilator reactions, particularly on painful stimulation, need special consideration. Some authors explain these phenomena by an irritative process around the sphincter nucleus (Behr [5]) or in the centripetal part of the light reflex arc (Sven Ingvar [8]). The latter author assumes that syphilitic meningitis irritates the pupillomotor optic fibers, inducing in this way tonic irritation of the sphincter nucleus and the sphincter muscle, respectively, so that the dilator impulses are ineffective. Yet it has already been pointed out by Wilson [10] that

26. Marburg, O.: Wien. Klin. **29**:235, 1903.
27. Wilson, S. A. K.: J. Neurol. & Psychopath. **2**:1, 1921.
28. Bumke, O.: Pupillenstörungen, Jena, Gustav Fischer, 1911.

miosis cannot be due to irritation or excitation, for it may continue for many years and is unaccompanied by any spasm of accommodation. Furthermore, one has to bear in mind that an irritative process in the subarachnoidal space acting on the centripetal part of the light reflex arc would always produce bilateral miosis on account of the semidecussation of the secondary optic pathways in the posterior commissure. Thus even unilateral stimulation of the superior quadrigeminal brachium could never produce the miosis and the loss of the dilator reaction in one pupil alone, as seen in the cases of unilateral Argyll Robertson pupil.

As to the theories that assume the destruction of the dilator pathways in the neighborhood of the sylvian aqueduct (Wilson,[10] Merritt and Moore [9]), it was shown in the present series of experiments that such lesions are not sufficient to abolish dilatation of the pupil on painful stimulation. The pupillary reaction in response to pain is retained even after severance of the cervical sympathetic and extirpation of the tectum mesencephali with the central gray matter around the aqueduct until close to the oculomotor nucleus (Gozzano [19]). Furthermore, in cases of the Argyll Robertson pupil the vestibular pupillary reflex (miosis, then mydriasis and finally hippus) may be abolished. This reaction is retained in paralysis of the dilator muscle (cervical sympathectomy), but destroyed by severance of the oculomotor nerve and is brought about by vestibular impulses that are carried from either labyrinth and its nuclei mainly by means of the homolateral and contralateral posterior longitudinal fasciculus (Spiegel [29]) to both sphincter nuclei [30]. Thus

29. Spiegel, E. A.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **25**:413, 1924; Wien. klin. Wchnschr. **38**:216, 1925.

30. Behr (in Wilbrand, H., and Sänger, A.: Neurologie des Auges, supplement. vol., Munich, J. F. Bergmann, 1927, p. 155) expresses doubt that the vestibular pupillary reflex is due to the conduction of vestibular impulses toward the sphincter nucleus through the posterior longitudinal fasciculus, because the vestibular impulses cause an increase of tonus in the nuclei of the external ocular muscles and could not cause in the same time dilatation of the pupil due to a lessened tonus in the sphincter nucleus. Yet it is well known (Topolanski, Bartels, Magnus and de Kleijn) that in vestibular nystagmus the external ocular muscles are in turn stimulated and inhibited, and alternate excitation and inhibition of the sphincter nucleus due to vestibular stimuli is therefore not strange. The assumption of a hypothetic pontile gaze center for the conduction of vestibular impulses to the ocular muscle nuclei seems unnecessary, since direct fiber connection from the vestibular nuclei into the posterior longitudinal bundle is well known (Leidler and others). Cases of reflex immobilization of the pupil in cases of tumor of the quadrigeminal bodies, as mentioned by Behr, cannot be used to answer questions of finer localization, as it is always uncertain how far the effect of the pressure reaches. Behr's questions concerning the light reflex in cases of lesions of the corpora quadrigemina and of the central gray matter around the aqueduct are answered by the experiments of Spiegel and Nagasaka. Thus the aforementioned findings in regard to the vestibular pupillary reflex are not invalidated by Behr's arguments.

the sphincter nucleus loses in the Argyll Robertson phenomenon its reactivity not only in response to impulses that reach this nucleus dorsally (optic impulses), but also the reactivity to impulses conducted to the nucleus ventrally (vestibular impulses).

One can hardly assume in analogy to Bumke's theory in regard to the optic fibers a selective degeneration of the vestibular reflex collaterals around the sphincter nucleus, because the action of the labyrinth on the external ocular muscles is retained in cases of the Argyll Robertson pupil at the same time that the vestibular pupillary reflex is lost. Furthermore, Spiegel observed cases of unilateral Argyll Robertson phenomena in which the vestibular pupillary reflex and the light reflex were both lost in the same eye. It is hardly conceivable that all optic and all vestibular reflex collaterals should degenerate around one sphincter nucleus, while the corresponding fibers around the opposite nucleus should remain intact. In order to prevent all reflex impulses from reaching the sphincter nucleus, a lesion must encircle this nucleus, dorsally as well as ventrally; yet it must be so small that it can remain limited on one side. This requirement seems to be easily met if the lesion is located at the junction between the cells of the sphincter nucleus and the various reflex collaterals that terminate here.

This leads to the fourth theory:

4. A lesion at the synapses around the cells of the sphincter nucleus, i. e., between the afferent and the efferent neurons of the light reflex arc (Spiegel [29]). This theory is first of all confronted with the question why the pupillary convergence reaction is retained and even increased in Argyll Robertson pupils, while the light reflex is abolished. The convergence reaction that is due to an irradiation from the convergence center (Perlia's nucleus) toward the sphincter nucleus is more resistant than the light reflex, as has already been pointed out by Bumke [28] (see also Behr [5]). Bumke stated that after instillation of atropine the light reflex of the pupil is extinguished sooner than the convergence reaction. In incomplete lesions of the oculomotor nerve the convergence reaction may be retained, although the light reflex is abolished (Bauer [23]). It is therefore conceivable that a lesion of the synapse prevents the optic reflex impulses from reaching the sphincter cells, while the more resistant impulses that radiate from the convergence center can still reach these cells. This of course explains only why the convergence reaction of the pupil is retained in the Argyll Robertson pupil; it does not explain why this reaction is often increased. Yet experiments of Sherrington [31] and Bremer [32] on the spinal cord have shown that a reflex center (e. g., the anterior horn cells) can become overexcitable after elimination of

31. Sherrington, C.: Quart. J. Exper. Physiol. **2**:109, 1909.

32. Bremer, F.: Ann. de physiol. **4**:756, 1928.

the main afferent impulses (e. g., by severance of the posterior roots of this side). Bremer, for instance, observed that vestibular impulses act more intensely on the anterior horn cells on the side where the posterior roots have been severed. Such "isolation" phenomena (Munk) are apparently due to the fact that the motor reflex center stores surplus energy when the main afferent impulses are shut off, so that the remaining impulses that reach the center find it in a state of hyperexcitability. In our case not only the retinal but also the other sensory and emotional impulses are shut off, and thus the convergence impulses find the sphincter center in a state of hyperexcitability, so that an increased convergence reaction may result.

This leads to an explanation of the miosis. If the centripetal impulses to a reflex center are shut off, the hyperexcitability of the "isolated" center in response to the remaining nervous and metabolic stimuli and the ensuing state of hyperexcitation may also induce an increased tonicity of the corresponding motor effector.

In experiments of Ranson,[33] for instance, a rigidity developed on the deafferented hindlimb. Another example can be observed in the vestibular apparatus. After extirpation of one labyrinth or severance of the eighth nerve a compensatory mechanism is established due to the development of a state of hyperexcitation in the vestibular nuclei on the side of the extirpation of the labyrinth (Spiegel and Démétriades[34]). This hyperexcitation balances the impulses from the remaining labyrinth, so that the nystagmus disappears, and it manifests itself after extirpation of the second labyrinth by Bechterew's compensatory nystagmus.

One can therefore conceive that a similar hyperexcitability and state of hyperexcitation develop in the sphincter center if the optic reflex impulses as well as the sensory and emotional inhibition are shut off. The corresponding sphincter muscle is then reached only by the constrictor impulses that are associated with convergence impulses and by tonic impulses from the sphincter center, but by no inhibitory impulses, so that miosis results. Thus the miosis can be explained as a consequence of the loss of reflex impulses that act normally on the sphincter center, and the assumption of an irritative process seems not necessary.

In the further progression of the miosis the development of a secondary contracture seems to play a part, as has already been assumed by Heddaeus, Behr[5a] and Redlich.[25] Such secondary changes of the sphincter muscle are suggested by the fact that the extremely miotic forms of Argyll Robertson pupil do not react to a toxin that paralyzes the peripheral end-apparatus, such as atropine. Observations of Levin-

33. Ranson, S. W.: J. Comp. Neurol. **52**:341, 1931.

34. Spiegel, E. A., and Démétriades: Arch. f. d. ges. Physiol. **210**:215, 1925.

sohn and Arndt [35] point in the same direction. The instillation of cocaine or pilocarpine into the conjunctival sac was in some cases of irregular miotic pupils effective only on some parts of the pupil, so that the irregularity persisted, apparently owing to partial secondary changes in the sphincter. No further explanation is needed for the fact that after the development of the secondary contracture excitation of the relatively weak dilator muscle by toxins or nerve impulses remains ineffective.

This theory explains the double-sided as well as the unilateral Argyll Robertson pupil and also the development of irregularities of the pupil. The transition from the Argyll Robertson pupil to the loss of all pupillary reactions is also comprehensible; it takes place when the injury to the synapses encroaches on the cell bodies of the sphincter center. Sometimes, particularly in the early stages of Argyll Robertson pupil, the light reflex can recover, e. g., after treatment with malaria (Clark [36]). Such a recovery could not be understood if the impairment of the light reflex were due to a degeneration of secondary pathways, because regeneration of degenerated central tracts does not occur. Yet a return to the normal state from a purely physicochemical change on the synapses around the cells of the sphincter nucleus seems possible, particularly in the initial stages. Finally, one cannot be astonished that the histopathologic methods have failed so far to detect the site of the lesion in Argyll Robertson pupils. New methods that enable one to study the synapses are needed to show definitely the changes that cause this phenomenon.

SUMMARY

Combined lesions of the posterior commissure and of the central pupillodilator tracts were produced in cats, and the effect of such lesions on the pupillary light reflex, on the response to painful and emotional stimuli and on the reaction of the pupil to atropine was studied.

It was found that unilateral severance of the posterior commissure induces on the side of the lesion a slight mydriasis and a diminution of the light reflex but not a loss of this reaction.

If this lesion is combined with that of the central pupillodilator pathways, slight miosis of the homolateral pupil results; the dilator response of the corresponding pupil to painful or emotional stimuli is diminished but not abolished; after the instillation of atropine into both eyes the miotic pupil dilates somewhat more slowly, but finally nearly the same extent of dilatation as that of the opposite pupil may be reached. Such combined lesions do not produce the picture of the Argyll Robertson pupil in cats, so far as the aforementioned reactions are concerned.

35. Levinsohn, G., and Arndt, M.: Deutsche Ztschr. f. Nervenh. **20**:397, 1901.

36. Clark, C. P.: Am. J. Ophth. **13**:946, 1930.

On the basis of these experiments and of former experiments of Spiegel the various theories of the Argyll Robertson pupil are discussed. The assumption of a lesion on the synapses between the afferent and the efferent parts of the pupillary light reflex arc seems to give the best explanation of all the symptoms of the Argyll Robertson pupil.

ABSTRACT OF DISCUSSION

DR. HARRY S. GRADLE, Chicago: I shall speak of one phase of the complexity known as the Argyll Robertson pupil, namely, the miosis. The size of the pupil in health is a result of a balanced condition between the sphincter muscle of the iris, the dilator muscle of the iris and the natural tonicity of the tissue of the iris. The first two of these factors are under nerve control and consequently variable, thus accounting for the continuous variations in the size of the pupil. When all external and psychic excitations are absent, as in sleep, the pupil is miotic, and the deeper the sleep the greater the miosis. But the miosis of the Argyll Robertson pupil has a different basis.

The authors have suggested that a lesion of the posterior commissure, combined with a lesion of the central pupillodilator pathways, produces a miosis, but not characteristic of that of the Argyll Robertson pupil. So they assume that a lesion on the synapses between the afferent and efferent parts of the pupillary light reflex arc offers the best explanation. But cannot the miosis be explained more readily by assuming a paresis of the sympathetics that stimulate the dilator fibers of the iris? Isolated nerve and muscle paresis is of frequent occurrence in neurosyphilis, as witnessed by the frequency of paresis of the abducens nerve. Is it not just as logical to assume a paresis of the dilator fibers of the iris, which would explain the miosis, the failure of the pupil to dilate completely under atropine and the irregularity of outline? To produce such a paresis the lesion would have to be located in the hypothetic dilator nucleus that occupies an unknown area somewhere in the immediate vicinity of the third nucleus. This would fit in with the lesion assumed by the authors to be lying in the synapses between the afferent and efferent parts of the pupillary light reflex arc.

DR. C. W. RUTHERFORD, Iowa City: The investigation of possible anatomic sites for lesions that may account for the Argyll Robertson pupil involves procedures of a highly technical character by skilled workers. Our sincere appreciation is due to all investigators who have contributed to the knowledge of the subject.

The occurrence of the unilateral Argyll Robertson pupil has caused confusion and impeded progress. The bilateral exhibition of the phenomenon has been produced in experimental animals by lesions in several locations near the anterior end of the cerebral aqueduct. When it is attempted to make any such location account for a unilateral exhibition, speculation is required as a support to experiment. The present authors have excluded one locality after another and then by ingenious reasoning have selected the most convenient and perhaps the most plausible and logical site for a lesion that can account for the Argyll Robertson pupil, whether in one eye or both, namely, the synapses between the afferent and efferent neurons in the reflex pathways for

pupillary contraction to light and the immediate vicinity of the third nerve nucleus for impairment of dilatation in reaction to atropine and to painful and emotional stimuli.

The authors start with an assumption of a lesion in and both dorsally and ventrally to the nucleus. They recognize the necessity of accounting for the preservation and even increased sensitiveness of the convergence reaction of the pupil. They quote authors to show that the pathway for contraction to convergence is more resistant to injury than the pathway for contraction to light. This relatively increased resistance may be due to a phylogenetic difference, to a more stable structural texture or to a relatively greater immunity to disease processes.

The Argyll Robertson pupil is regarded as permanent. The authors speculate on the hyperexcitability of the nucleus to convergence impulses. In time there should be some change somewhere that would compensate for the hyperexcitability and its influence on function; for instance, extirpation of one labyrinth produces a nystagmus which will eventually disappear.

The paper states that "Spiegel observed cases of unilateral Argyll Robertson pupil in which the vestibular pupillary reflex and the light reflex were both lost in the same eye." These reflexes are mediated through quite different systems, and the defects probably coexist because of their coincidental occurrence as results of the same disease rather than because of intimate neural connections.

The authors discuss secondary contractions in the sphincter of the iris and irregularity in the contour of the pupil; sometimes only parts of the iris respond to atropine, cocaine and pilocarpine or to nerve impulses. This so-called secondary contraction, which affects part or all of the sphincter, might be due to a permanent failure of blood supply or to atrophy of the iris rather than to a permanent contraction of otherwise healthy tissue such as occurs in striped muscles. This interpretation would be plausible if such an iris were found friable or fragile on attempted iridectomy.

The lid-closing reflex, Gifford's sign, does not seem to have been considered to any great extent in any studies on this subject. It might be advantageous to include it in future investigations.

Syphilis is a disease that acts diffusely. The lesions due to it are not identical in nature or probable effect with those produced by a sterile knife or cautery in normal cats, because in the latter the diffuse effects of disease are not present.

Fifty early or fairly mild cases of neurosyphilis were studied clinically; in all of them the central fields and blindspots could be plotted with a 1 mm. white object at a distance of 1,200 mm. on the Bjerrum screen. The cases were studied with reference to pupillary and field defects.

It was found that Argyll Robertson, oval and irregular pupils and anisocoria were preponderatingly associated; that, with field defects, normally acting and Argyll Robertson pupils were about equally present, and that there was a progressive incidence of Argyll Robertson pupils in meningovascular syphilis, paresis, tabes and mixed types of neurosyphilis. The mixed types illustrate how diffusely syphilis can affect the central nervous system. There was only one case in which the Argyll Robertson pupil was unilateral. It is probable that all patients who have

unarrested neurosyphilis will eventually have bilateral Argyll Robertson pupils, but it may be confidently expected that one eye will become involved before the other. Possibly the unilateral phenomenon has received more consideration in laboratory studies than it deserves.

Dr. Ernest A. Spiegel, Philadelphia: Dr. Gradle brought out the interesting question whether miosis might be due to a lesion of the dilator tract. I believe that our experiments and those of my co-worker, Dr. Gozzano, have shown that any lesion of the central or peripheral part of the dilator tract is not sufficient to produce all the symptoms of the Argyll Robertson pupil. It might produce a miosis, but such a lesion does not prevent development of the dilator reflexes due to painful stimuli. Therefore, I believe that a lesion in the dilator tract is not sufficient to explain the loss of the dilator reactions.

The light reflex and the vestibular pupillary reflex have a common path between the synapses surrounding the cells of the sphincter nucleus and the motor effector. If both reactions are abolished, it seems logical to assume the presence of a lesion in a part of this common path. If permanent failure of the blood supply of the iris could play a part in the genesis of the extreme miosis, it should be possible to show such changes of the blood supply by histologic examination.

It is true that the lid-closing reflex should be studied more.

I agree that syphilis plays an important part in the majority of cases. We were particularly interested in the cases in which the condition was unilateral, although they are relatively rare. But any theory of the Argyll Robertson pupil must explain all its features, whether they are rare or not.

BIOCHEMISTRY OF THE LENS

VI. MINERAL METABOLISM IN THE NORMAL AND IN THE CATARACTOUS LENS

JAMES E. LEBENSOHN, M.D.
CHICAGO

The effect of age on the metabolism of the lens can be studied by two diverse methods: Comparisons may be made of lenses representing different age periods of a species, or of successive zones of the adult lens. Thus data on the quantitative distribution of the lens proteins in whole bovine lenses from animals of different ages (fetuses, calves, steers, old cows)[1] parallel those derived from analysis of the zones of the adult lens from without inward.[2]

Just as a contrast of the biochemical findings in the cortex and nucleus of the normal lens gives an insight into the aging process, so a further comparison of corresponding data on cataractous lenses may indicate to what extent age influences cataractous degeneration.

Certain mineral changes are characteristic of cataract, namely, a loss of potassium and a gain in sodium and calcium. Since the osmotic pressure of a tissue is dominantly determined by its mineral content, and since anions and cations are in balance, osmotic status may be simply represented by a calculation of the ionic concentration of diffusible base in terms of milliequivalents[3] present per liter of available water.[4] On the basis of these theoretical considerations I calculated that the osmotic pressures of the normal and the cataractous lens were essentially the same,[5] a conclusion that Nordmann[6] recently verified experimentally by careful cryoscopic measurements. I interpreted the gain in sodium as merely effecting an osmotic compensation for the loss

From the Department of Ophthalmology, Northwestern University Medical School.

1. Lebensohn, J. E.: Unpublished data.
2. Krause, A. C.: The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934, p. 212.
3. In univalent ions, such as sodium and potassium, the "equivalent" is identical with the atomic weight, one milliequivalent being one one thousandth of the atomic weight.
4. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1932, vol. 1, chap. 15.
5. Lebensohn, J. E.: Water Equilibrium in the Normal and Cataractous Lens, Am. J. Ophth. **16**:1062, 1933.
6. Nordmann, J.: Etudes physico-chimiques sur le cristallin normal et pathologique, Arch. d'opht. **52**:78, 1935.

of potassium. Protein degeneration determines the loss of potassium, and the gain in calcium is a secondary phenomenon which runs parallel thereto.[7]

In the accompanying table, the average of the determinations of the sodium, potassium and calcium content of the cortex and nucleus of normal adult bovine lenses and of human cataractous lenses, respectively, has been figured as the percentage of ash, the percentage of dried weight and the milliequivalents per liter of available water. The lens material was dried to constant weight over calcium chloride and dry-ashed in platinum crucibles in an electric furnace at 400 C. Sodium was determined by the uranyl zinc acetate method as modified by Salit,[8] and potassium and calcium by the methods of Kramer and Tisdall.[9] The water content of the cataracts was ascertained from intracapsularly

Mineral Metabolism of Lens

Material	Water/Dry Weight	Percentage of Ash in Relation to			Percentage of Mineral in Ash			Percentage of Mineral in Dried Weight			Milliequivalents per Liter of Available Water			Potassium/Sodium, Milliequivalent Values	Potassium/Calcium, Ash Values
		Dry Weight	Wet Weight	Water Content	Sodium	Potassium	Calcium	Sodium	Potassium	Calcium	Sodium	Potassium	Total		
Normal ox lens															
Cortex....	2.33	2.66	0.89	1.04	10.7	30.2	1.1	0.2950	0.8213	0.028	54.5	90.1	144.6	1.65	27.5
Nucleus...	1.04	1.31	0.64	1.26	16.4	37.5	...	0.2154	0.4923	.....	90.0	120.6	210.6	1.34	....
Human cataract															
Cortex....	3.00	2.87	0.72	0.96	25.7	17.8	2.3	0.7348	0.5068	0.065	106.6	43.2	149.8	0.41	7.7
Nucleus...	1.50	2.12	0.85	1.41	28.0	15.0	4.7	0.5806	0.3117	0.097	168.3	53.2	221.5	0.32	3.2

removed lenses—that of the nucleus directly and that of the cortex by deduction from the data on whole lenses.

In the normal lens, the percentage of ash in the cortex is twice that in the nucleus when the ashes are compared with the respective dry weights (table), but only about one-third more when they are compared with the fresh weights, and but five-sixths as much in relation to the water contents. In the cataractous lens, the table discloses that there is a marked increase in the ash of the nucleus, so that the cortex contains only about a third more in respect to the dry weight, six-sevenths

7. Salit, P. W.: Calcium Content and Weight of Human Cataractous Lenses, Arch. Ophth. **9**:571 (April) 1933.

8. Salit, P. W.: A New Triple Acetate Method for Sodium Determination in Biological Materials, J. Biol. Chem. **96**:659, 1932.

9. Kramer, B., and Tisdall, F. F.: The Direct Quantitative Determination of Sodium, Potassium, Calcium and Magnesium in Small Amounts of Blood, J. Biol. Chem. **48**:223, 1921. Tisdall, F. F., and Kramer, B.: Methods for the Direct Determination of Sodium, Potassium, Calcium, and Magnesium in Urine and Stools, ibid. **48**:1, 1921.

as much in regard to the wet weight, and but two-thirds as much in relation to the water content.

According to the data submitted here, the entire calcium content of the normal lens appears to be in the cortex. In the belief of Wessely,[10] Kranz [11] and others, the calcium of the normal lens is probably in colloidal solution. The nuclear ash, however, is shown to contain the larger portion of both sodium and potassium. This finding may account for the greater basicity of the nucleus, noted by Buglia,[12] and possibly for its more alkaline iso-electric point. That the nucleus is richer in potassium than the cortex has been previously reported by Burdon-Cooper [13] from spectroscopic observations.

In cataract there is a decrease in potassium with an increase in sodium, which, as my analyses indicate, affects both cortex and nucleus. The loss of potassium in both instances is of the same proportion in relation to the dry weight. For, thus calculated the loss of potassium in the cortex is $\frac{0.8213-0.5068}{0.8213}$ or 38.3 per cent, while the loss of potassium in the nucleus is $\frac{0.4923-0.3117}{0.4923}$ or 36.7 per cent.

In the cataractous lenses, the potassium plus sodium totals for cortex and nucleus, when calculated in ionic concentrations per unit of water, reach similar figures as compared with the respective normal totals (table), indicating again that no essential change has taken place in their osmotic relationships. Since the nucleus must be in osmotic equilibrium with the cortex, the higher value of the milliequivalents in both the normal and the cataractous nucleus indicates that about one fourth of its sodium and potassium is in indiffusible combination with the protein therein.

Using the ratio of potassium to sodium as an index of tissue vitality —a conception elaborated by Gérard [14]—one finds from the calculations of milliequivalents a progressive decrease in the quotient from normal cortex (1.65) and normal nucleus (1.34) to cataractous cortex (0.41) and cataractous nucleus (0.32).

The ratio of potassium to calcium shows a characteristic decrease of quotient with the advance of cataract. Whereas the normal cortex gives the quotient as 27.5, that of cataractous cortex is 7.7 and that of

10. Wessely, K.: Zwei kleine Beiträge zur Pathologie der Linse, Arch. f. Augenh. **91**:158, 1922.

11. Kranz, H. W.: Die polarisationsmikroskopische Untersuchung der kataraktös getrübten Linse, Arch. f. Ophth. **118**:571, 1927.

12. Buglia, G.: Sur la réaction chimique actuelle du cristallin en condition de transparence normale et dans l'opacité, Arch. ital. de biol. **76**:1, 1926.

13. Burdon-Cooper, J.: The Pathology of Cataract, Tr. Ophth. Soc. U. Kingdom **53**:401, 1933.

14. Gérard, P.: Teneur en potassium et en sodium des différents organes d'un chien, Compt. rend. Acad. d. sc. **154**:839, 1912.

cataractous nucleus 3.2. It is noteworthy that Galeazzi [15] found no essential difference in the potassium-calcium quotient of sclerotic cataracts as compared with cortical types.

Histologic studies (Wessely,[10] Kranz [11]) have shown that in cataract the calcium is principally deposited in the nucleus. By the use of Gömöri's [16] specific stain for calcium I have personally confirmed this observation. My chemical analyses corroborate the correctness of these observations, calcium forming 2.3 per cent of the ash in the cataractous cortex and 4.7 per cent of that in the cataractous nucleus. Krause [17] holds that calcium tends to be deposited in the albuminoid as also in or around other inert insoluble substances, especially if the metabolism of the adjacent surrounding tissue is low. Another consideration that favors the deposition of calcium in the nucleus is its somewhat more alkaline reaction. Moreover, as Fischer [18] pointed out, potassium is antagonistic to calcium in its colloidal chemical action, which may account for the fact that calcium increases in the lens as potassium decreases. Mackay, Stewart and Robertson [19] found that calcium accounted for 1 per cent of the ash of normal human lenses; but in traumatic cataract it accounted for 1.3 per cent; in immature cataract, for 2.3 per cent, and in mature cataract, for 5.5 per cent. Evans and Kern [20] reported that whereas in the normal dog's lens calcium was 1.5 per cent of the ash, in the cataractous lenses of parathyroidectomized dogs it increased to 28.7 per cent. This disproportionate deposition of calcium is probably due to involvement of a special factor consequent to the loss of the parathyroid hormone.

Fischer [18] maintained that all tissues have a distinctive mineral content, and that water-rich and water-poor tissues differ characteristically in this respect. The lens has the least water content of any protein tissue in the body and is particularly high in potassium and sulfate ions and low in sodium and chloride ions. Fischer also noted that in the aging lens potassium increases and sodium decreases, but this increase in potassium is mostly in the nucleus.

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15. Galeazzi, C.: Cattaratta ed equilibrio calcio-potassico, Rossegna ital. d'ottal. **2**:863, 1933.

16. Gömöri, G.: Der mikrotechnische Nachweis unlöslicher Kalksalze in den Geweben, Virchows Arch. f. path. Anat. **286**:682, 1932.

17. Krause,[2] p. 196.

18. Fischer, F. P.: Der Mineralbestand des Auges, Arch. f. Augenh. **107**:295, 1933.

19. Mackay, G.; Stewart, C. P., and Robertson, J. D.: A Note on the Inorganic Constituents of Normal and Cataractous Human Crystalline Lenses, Brit. J. Ophth. **16**:193, 1932.

20. Evans, E. I., and Kern, R.: Relation of Parathyroid Glands to Cataract, Am. J. Ophth. **14**:1029, 1931.

In micro-incineration of the lens, central and peripheral areas are definitely demarcated (Policard and Bonamour,[21] Zeidler[22]). In the nucleus the ash is more abundant than that of the cortex and of different aspect.

CONCLUSIONS

1. The nucleus contains normally a greater proportion of basic ions than the cortex.

2. The calcium accumulation in cataract is mostly in the nucleus.

3. About one fourth of the potassium and sodium in the nucleus is apparently indiffusibly bound to the protein.

4. Separate analyses of cortex and nucleus confirm the view that the gain in sodium in cataract occurs simply to compensate osmotically for the loss in potassium.

5. The ash of the cortex is more than that of the nucleus when the ashes are compared with the respective dry weights, but less in relation to the water contents.

6. Cataractous degeneration, so far as may be determined from the mineral analyses, is a diffuse process; in the cataracts that come to operation cortex and nucleus seem about equally involved.

Prof. S. R. Gifford of the Department of Ophthalmology and Prof. C. J. Farmer of the Department of Biochemistry of Northwestern University Medical School extended many courtesies and suggestions.

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21. Policard, A., and Bonamour, G.: Etude par micro-incinération de la cornée, du cristallin et des formations iridociliares de l'oeil humain, Bull. d'histol. appliq. à la physiol. **10**:362, 1933.

22. Zeidler: Demonstration spodographischer Präparate, Ber. d. deutsch. ophth. Gesellsch. **107**:295, 1933.

HEREDITARY CATARACT OF THE SENILE AND PRESENILE TYPES

F. VINSONHALER, M.D.
Dean of the University of Arkansas School of Medicine
AND
K. W. COSGROVE, M.D.
Chief of the Department of Ophthalmology, Baptist State Hospital
LITTLE ROCK, ARK.

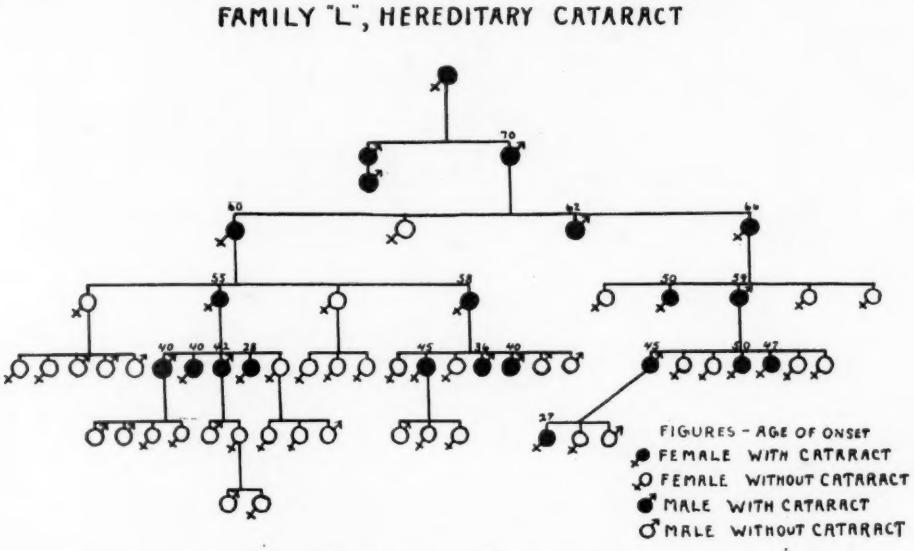
Heredity has been recognized for years as a factor in the development of presenile and senile cataract. The family reported on in this article adds to the weight of evidence in support of this fact.

The Loy family, whose genealogical tree is represented by the chart, have all resided in Arkansas in or about Hot Springs. All the members of the family, with the exception of those in the first generation, have been seen by one or by both of us. The cataracts were of the cortical type. Maturity was reached in from six months to three years after they were first noticed. The age of development of cataract has varied from over 70 in the second generation to 27 in the sixth generation. The members of the family in the fifth and sixth generations in whom cataract has not developed have not attained the age of expectancy for that generation. Ten of the children of cataractous parentage of the fifth and sixth generations have been examined with the corneal microscope, and no evidence of any pathologic process can be seen in the lens. However, two patients of the fourth generation in whom cataract later developed were examined and no changes were found in the lens twelve and eighteen months, respectively, before opacities appeared. The blood picture was entirely normal in all cases. The red cell count was above 4,000,000, and the white cell count varied between 5,000 and 7,000. The Wassermann reaction of the blood was negative. The urine was normal. The members of the family are healthy, live to a normal expectancy of life and are apparently fertile.

The cataracts have been removed by one of us, except those of the members of the first and sixth generations. The changes in the lenses of the patients of the sixth generation are in very early stages. The extracapsular operation with iridectomy was done in all cases, and needling of the posterior capsule was necessary before a clear pupil was obtained. The vision obtained two months after operation has been from 20/20 to 20/30, and the patient has been able to read the Jaeger test type no. 1 except in the case of one member of the third generation. The result in this case was 20/30 in the first eye which was operated

on. Twelve years later a large corneal ulcer developed in this eye. This resulted in a large leukoma, covering the entire cornea. The second eye was then operated on, when the cataract was thirteen years overmature. The vitreous had many opacities, and the best vision that could be obtained was 20/100, with ability to read the Jaeger test type no. 3.

In 1905 Nettleship[1] made a very complete report on hereditary cataract from a résumé of the literature and from his own records. This report covered 122 families and 375 persons with cataract of the presenile and senile types. In 35 of these families the cataracts were in siblings; in 47, in only two generations; in 31, in three generations, in 5, in four generations, and in 4, in five generations. Since this report



Genealogical chart, showing the incidence of cataract in the Loy family.

the incidence of cataract in 6 families has been reported by Rowan and Wilson,[2] Parker[3] and Casey Wood.[4]

Nettleship[1] laid down six rules pertaining to the occurrence of cataract in these families. It is of great interest to note that the Loy family follows the majority of these rules. They are as follows, to which are added comments:

1. The descent is direct. No generations are skipped. Children of a member of the family not having cataract can feel assured that one will not develop. The members of the Loy family whose parents

1. Nettleship, E.: Roy. London Ophth. Hosp. Rep. **17**:389, (Oct.) 1905.
2. Rowan, John, and Wilson, J. A.: Brit. J. Ophth. **5**:64, 1921.
3. Parker, W. R.: J. Michigan M. Soc. **15**:188 (April) 1916.
4. Wood, Casey: Ophth. **15**:142 (April) 1906.

showed no lenticular changes did not have cataract. Fifty per cent of these have passed the age of expectancy for their generation.

2. If all children in one family have cataract, the liability of the next generation is increased.

3. Transmission from like sex to like sex is most common. In the family reported on here heritage is from unlike sex in 12 instances and like sex in 9.

4. Cataract tends to appear earlier in life in succeeding generations. In the members of the Loy family in the second generation cataract developed at the age of 70, and in each succeeding generation the age of onset decreased one decade, the members of the sixth generation showing lenticular opacities at the age of 27. It is feasible to presume that if this rule continues in the same ratio the members of the tenth or eleventh generation might be born with cataract.

5. The age of onset is approximately the same in persons of the same generation. (This is of interest to the members of that generation in that they can know what to expect.)

6. The families with cataract are not affected as to fertility, health or longevity.

HERPES CORNEAE

WITH SPECIAL REFERENCE TO ITS TREATMENT WITH STRONG SOLUTION OF IODINE

TRYGVE GUNDERSEN, M.D.
BOSTON

In New England dendritic keratitis appears to be the most frequent corneal infection recognizable on an etiologic or morphologic basis. During the past forty-two months 221 patients with this disease have been observed and treated personally by me. These patients comprise the majority of all those with corneal infection seen during this period in the outpatient department of the Massachusetts Eye and Ear Infirmary. The frequency of dendritic keratitis in comparison with that of other well recognized conditions of the cornea is shown in table 1.

TABLE 1.—*Incidence of Various Types of Keratitis During Forty-Two Months*

Total number of new patients in the outpatient department	41,733
Dendritic keratitis	221
Interstitial keratitis (syphilitic and nonsyphilitic)	184
Serpent ulcer	97
Sclerokeratitis	80
Phlyctenular keratitis	52
Gonorrheal ulcer (all ages)	27
Disciform keratitis	18

Trophic or senile marginal ulcers and those attributable to rosacea have been omitted from consideration, since their classification is not sufficiently clear for statistical study.

The 221 cases of dendritic keratitis were studied intensively. When necessary, special consultations were given by the medical, dental, urologic and rhinolaryngologic departments of the Massachusetts General Hospital. Patients requiring special study or hospital care were admitted to the wards of the Massachusetts Eye and Ear Infirmary.

INOCULATION OF RABBITS

Inoculation of a rabbit was carried out in each case during the first two years of this study, but later only in those cases in which there was doubt as to the diagnosis. The method used in inoculation was that

From the Howe Laboratory of Ophthalmology, Harvard University, and the Massachusetts Eye and Ear Infirmary.

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described by Löwenstein [1] and Grüter,[2] namely, the immediate transfer of scrapings from the dendritic ulcer to four or five parallel scratches in the corneal epithelium of the rabbit. Dendritic keratitis becomes evident in from twenty-four to seventy-two hours, the time of its appearance probably depending on the activity of the virus. A rabbit with normal corneas was used for every inoculation. The opposite cornea of the same rabbit was often used if the second inoculation was done before immunity could have developed, that is to say, within one week after the first inoculation.

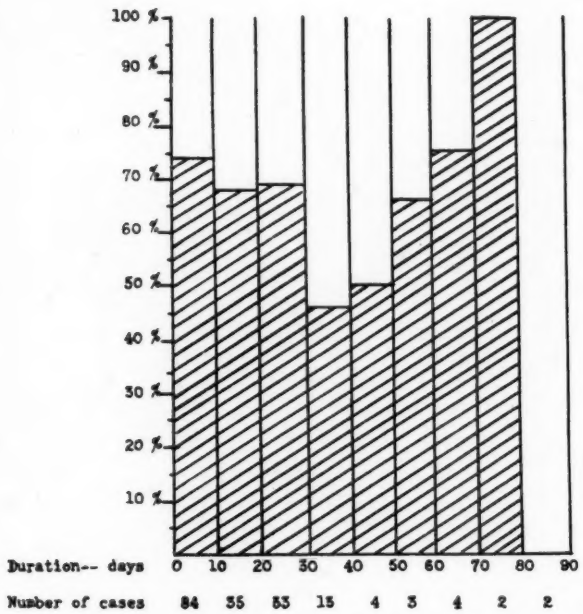

Chart 1.—Relation of the number of positive reactions to tests on rabbits to the duration of the disease. The total number of tests was 180; the number in which the reaction was positive was 113, or 63 per cent.

The virus of herpes may remain demonstrable in a dendritic ulcer for several months. Of 28 cases in which the condition had been active for more than thirty days the inoculations gave positive results in 15, or 54 per cent. In 2 cases the virus was demonstrable after the sixtieth day of the disease, and in 2 other cases even after the seventieth day. From chart 1 it appears that no relationship between positive results of tests on rabbits and the duration of the disease can

1. Löwenstein, A.: Uebertragungsversuche mit dem Virus des fieberhaften Herpes, Klin. Monatsbl. f. Augenh. **64**:15, 1920.

2. Grüter, W.: Experimentelle und klinische Untersuchungen über den sogenannten Herpes corneae, Klin. Monatsbl. f. Augenh. **65**:398, 1920.

be reasonably proved except for the first ten day group in which the number of positive results is significantly in excess of what is expected. If the disease appears clinically to be active herpetic keratitis, there is slightly less chance of proving the presence of the virus in a case of long than in one of short duration.

A total of 623 inoculations was made, in 180 of which the scrapings were from patients with definite dendritic keratitis. To make sure that atypical cases of herpetic keratitis were not overlooked, all varieties of superficial punctate keratitis, keratitis nummularis, keratitis epithelialis, epithelial and endothelial dystrophies, traumatic relapsing keratitis, metastatic gonorrheal keratitis, bullous keratitis, herpes zoster keratitis and disciform keratitis were tested by inoculation of rabbits. Nevertheless, the presence of the virus of herpes was demonstrable only in cases of dendritic keratitis and in a few cases of disciform keratitis. Not a single case of vesicular herpetic keratitis, described in textbooks as an acute eruption of many fine vesicles distributed over the cornea, has been seen by me in the course of this study. In 37 per cent of the 180 cases of dendritic keratitis, the results of inoculation were negative, although the clinical picture was typical of the disease. This failure to demonstrate active virus suggests its death in the human cornea, its migration into the deeper corneal layers or some unexplained technical fault. Natural immunity of adult rabbits to the virus is unknown.

SEX AND AGE INCIDENCE

The sex incidence is remarkable. The disease appears about two and one-half times as frequently in adult males as in adult females. The adult series includes 126 males and 52 females. However, this disparity disappears below the age of 16 years. In this early age group, in a total of 46 cases there were 23 males and 23 females.

The age at which the first infection appears became of special interest very early in this study. It was astonishing how often dendritic keratitis in infants and in children was mistaken for interstitial keratitis, and even more frequently for phlyctenulosis. Therefore, inquiries were made in every case as to the age of the patient when the first attack was experienced. Division into age groups has been made (chart 2).

From this chart it appears that the incidence of the primary attack of dendritic keratitis is higher in the first two five year periods of life than in any other period. Later in life the incidence corresponds fairly closely to the curve representing the distribution of the population in Massachusetts in various age groups.

SEASONAL VARIATIONS

Seasonal variations are interesting. The month of onset has been recorded in each case. It is generally believed that the greatest incidence occurs during months when infections of the upper respiratory tract

are most common. This has not been definitely borne out in the present study, although the lowest incidence does appear to be in the months of September and October.

INCIDENCE OF OTHER ASSOCIATED INFECTIONS

Colds, grip and infections of the upper respiratory tract accompanied the majority of attacks in this group of patients. This association was observed 118 times in 190 attacks, or in 62.1 per cent. Care was exercised not to be misled by the patient's history of nasal discharge due to lacrimation from the inflamed eye. These associated diseases had no exact chronological relationship to the onset of the keratitis. Little

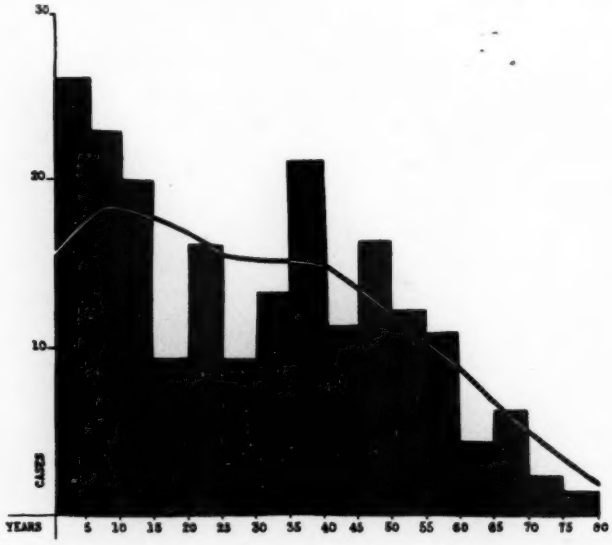

Chart 2.—Incidence of the primary attack of dendritic keratitis according to age group. The black columns represent the distribution of cases in various age groups; the curve, the distribution of the population in Massachusetts in age groups.

information could be obtained regarding the importance of malaria. Two patients gave histories of the disease, although dendritic keratitis was not present at the time of the malarial attacks. One patient with dementia paralytica, who had been infected with malaria therapeutically, had a severe dendritic keratitis during the height of the fever. In only 1 case of active pulmonary tuberculosis was dendritic keratitis found associated.

OCCURRENCE OF HERPES FEBRILIS ELSEWHERE

Herpes febrilis appeared elsewhere on the face 76 times in 203 attacks, or in 37.4 per cent of the cases in which this observation was recorded. A great many more patients in the series were subject to cold sores, although no association with the keratitis was obvious. On

the other hand, it was not uncommon for patients to deny any previous existence of herpes simplex by sign or symptom. Apparently the virus of herpes can make its appearance on the cornea alone without ever having involved any other part of the body.

TRAUMA

One patient was seen in the clinic with a foreign body in the cornea four days before the diagnosis of dendritic keratitis was made. Another (case 7) was seen in the clinic with three small corneal abrasions, presumably due to trichiasis; five days later there were three primary

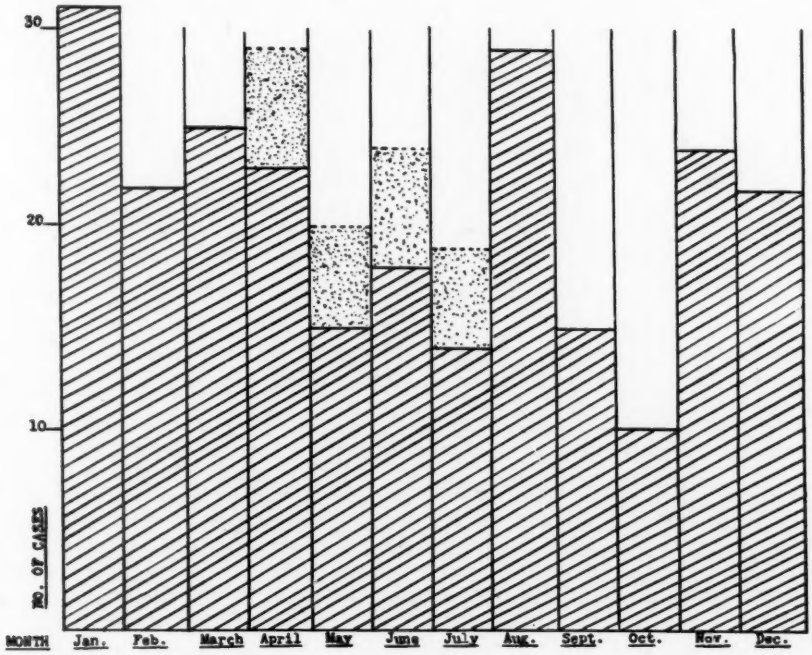

Chart 3.—Seasonal incidence of dendritic keratitis in 245 cases. The columns of diagonal lines represent the actual number of cases; the dotted areas, the theoretical correction from April to August 1935.

dendritic figures in the identical areas of the cornea in which these abrasions had been previously described. In a total of 250 attacks of dendritic keratitis observed, there was a history of possible injury in 24. Actual injury was considered to be probable in 13 of these, that is, in cases 3, 4, 5, 6, 7, 9, 10, 11, 13, 15, 17, 22 and 23 listed in table 2. Three of these, cases 13, 15 and 22, are of special importance because there was a definite injury closely preceding the first attacks which these patients had experienced. It may be concluded, therefore, that preceding trauma was probable in 5.2 per cent and possible in 9.6 per cent of the 250 attacks.

Foreign bodies of the cornea were found some time after an attack of dendritic keratitis more frequently than before. From the corneas of 2 patients foreign bodies were removed after the patient had been observed for several weeks, and from the cornea of 1 patient, after the disease had subsided completely.

The possibility of trauma playing a part in the etiology of dendritic keratitis raises the question whether the disease is of exogenous or of endogenous origin. The fact that a rabbit's cornea can be inoculated

TABLE 2.—*Data on Cases of Dendritic Keratitis Following Injury*

Case	Name	Age	Sex	Comment
1	Adams	28	M	Piece of glass removed by family physician 1 day before onset; also had grip at onset, with cold sores
2	Barrett	21	M	Coal dust blew in eye at onset; had severe cold 2 weeks before onset
3	Blake	77	M	Foreign body of cornea removed in clinic 4 days before diagnosis of dendritic keratitis was made; also had chronic bronchitis
4	Buonanno	21	M	Concrete splashed in eye at onset
5	Cucchira	39	M	Liquid shoe cement splashed in eye at onset
6	Cunnane	34	M	Piece of glass from exploding bottle struck eye at onset; had cold sores at onset
7	Donehey	60	M	Trichiasis with three small corneal abrasions; treated in clinic 5 days before diagnosis of dendritic keratitis was made
8	Fransen	41	M	Questionable injury—details unknown; had a cold sore at onset
9	Homer	32	M	Foreign body removed by family doctor 3 days after onset; had a cold with cold sores at onset
10	Howden	68	M	Branch of tree struck eye at onset
11	Lord	71	M	Ether anesthesia day before onset
12	Luzio	54	M	Foreign body removed by laborer on day of onset
13	Magnell	34	M	Linotype struck eye at onset of first attack 2 years previously; no injury associated with recurrence
14	Manning	38	M	Foreign body of eye removed by drug store clerk day after onset
15	Meighan	57	M	Accidental burn of eye with tincture of iodine 2 years before onset; had a cold at onset
16	Morris	14	M	Dirt removed from eye by friend one day before onset; had cold sores at onset
17	Movello	53	M	Chip of stone struck eye 5 days before onset
18	O'Donnell	42	M	Particles of rust fell into eye at onset; 10 days later eye again injured by visor of cap; had cold sores at onset
19	Parker	40	M	Exposure to fur cleansing fumes day before onset; had a cold with cold sores at onset
20	Sawyer	34	M	Foreign body of eye removed by wife 2 days after onset; had a cold sore at onset
21	Sherlock	35	M	Foreign body removed from eye by friend 3 days after onset
22	Soper	33	M	Foreign body of cornea removed 4 years ago; eye was very sore for 7 days at that time
23	Wassermann	17	M	Second degree sunburn of face day before onset; had cold with cold sores at onset
24	Whitcomb	65	M	Dust blew in both eyes day before onset

with the virus indicates that the disease can be exogenous, but does not prove that it is always so. A priori, the assumption seems reasonable that when the virus is present and the individual at the time susceptible, trauma to the eye, even if slight, increases the chance of corneal infection. This assumption, moreover, is consistent with the fact pointed out previously, that the disease is two and one-half times more frequent in males than in females over 16 years of age, since males are more exposed to trauma. The fact that trauma seldom results in the disease may be explained by the assumption that the presence of the virus and the susceptibility of the individual seldom coexist. But definite evidence for trauma as even a contributory cause is lacking, since (1) the disease

closely followed definite injury to the cornea only twice in 250 attacks; (2) at the most a history of injury was obtained only 24 times, and (3) the greater frequency in males still exists when all cases with a history of trauma are excluded from consideration. The most that can be said is that when the disease closely follows definite trauma it is probable that the trauma is the exciting cause. It is to be noted that the question of the relation of trauma to herpes of the skin presents difficulties similar to those in the case of herpes of the cornea.

HYPESTHESIA

A strong impression that hypesthesia develops during the attack rather than before results from this study. The reduction in corneal sensibility seems definitely proportional to the amount of corneal involvement and the total duration of dendritic keratitis in any given eye. Several patients with primary attacks have been seen who have had normal or even increased sensibility during the first few days of the attack and subsequently marked hypesthesia.

Corneal sensibility can be tested most accurately by the careful use of Frey hairs. However, these hairs are not practical for clinical use. A carefully prepared wisp of cotton with the terminal fibers cut off squarely leaving a stimulating surface area of about 0.25 sq. mm. is satisfactory. Even more useful has been found the subjective test of dropping a slightly irritating collyrium, such as 1 per cent phenacaine, into both eyes of the patient, preferably into the affected eye first. In the vast majority of cases it will be perfectly obvious which eye is hypesthetic. The patient will close the normal eye and complain of a great deal more discomfort in it than in the involved eye.

In most cases the hypesthesia is obvious, particularly after the first week of the disease. In no other corneal inflammation with the exception of neuroparalytic keratitis does one regularly see so much conjunctival congestion, corneal infiltration and ulceration associated with so little feeling of irritation.

TREAMENT

The primary purpose of this investigation was to evaluate the merits of a very old and well known therapeutic measure for dendritic keratitis, namely, the use of strong solution of iodine. This study could not be made on the corneas of rabbits infected with herpes simplex. It was found that if iodine was applied to the corneas of these animals more scarring resulted in every rabbit than if the disease was left untreated. The reason for this is perhaps to be found in the fact that in the rabbit there is no structure analogous to Bowman's membrane. It is therefore possible that strong solution of iodine has a much greater penetration in the cornea of the rabbit than in the cornea of man. The first patients with dendritic keratitis were treated cautiously, great care being exercised not to allow the strong solution of iodine to spread beyond the

ulcer onto the normal epithelium surrounding it. Some of the results obtained were not wholly satisfactory. It was assumed that not enough iodine had been applied, and with increasing boldness the solution was deliberately applied to the surrounding area. It soon became evident that the corneas of these patients healed with no more scarring than did those for which more caution had been exercised. This led to the application of strong solution of iodine to the entire cornea in cases in which the dendritic figure was widely spread. This procedure of total iodization was employed in 47 attacks. In the remaining 113 attacks, the solution of iodine was applied only to the ulcer and the immediately surrounding area. It is believed that with the exception of a few complicated cases described subsequently, iodine has not added to the amount of corneal opacity resulting from the disease, for in no uncomplicated case has the ultimate visual acuity been below that which was present before the treatment.

Strong solution of iodine (U. S. P.), which contains 7 per cent iodine and 5 per cent potassium iodide in alcoholic solution, was used. Applications were made with a semidry firmly rolled toothpick swab the cotton-swathed tip of which measured approximately 1 cm. in length and 4 mm. in diameter. With the eye thoroughly cocainized and the lids held widely apart, the swab, thoroughly moistened with the solution of iodine, was rolled over the area from three to five times, or until a deep chestnut-brown color was obtained. Further penetration by the solution of iodine was then stopped by again dropping cocaine solution over the cornea. Insoluble cocaine iodide is formed, the color of which is much lighter than that produced by the action of iodine alone on corneal epithelium. If sufficient iodine is applied, the lesion becomes a bright orange yellow. This blanches within two hours, leaving the epithelium white, opaque and necrotic. After twenty-four hours the cornea is entirely denuded of epithelium in the area treated, and a smooth glistening Bowman membrane can be seen, outlined by fresh epithelium growing from the periphery. Complete reepitheliation then occurs rapidly.

With the exception of a few nervous patients and a few others with only a slight degree of corneal hypesthesia, it was found advisable to admit to the hospital only those patients who required iodization of more than one half of the corneal surface. Pain is usually not distressing for more than four hours after treatment. Applications of iced compresses were employed in all cases, and often 0.600 Gm. of acetylsalicylic acid and 0.030 Gm. of codeine were administered by mouth. When the pain was unusually severe, a hypodermic injection of 0.015 Gm. of morphine was given.

The use of an eye cover was not found to influence the rapidity of corneal healing and was used only when the patient's comfort seemed to require it. Mydriatics were not ordinarily employed unless cells in

the aqueous or other evidence of intra-ocular inflammation could be detected with the slit lamp. Either scopolamine or atropine was used indifferently, the former in a 0.25 per cent solution and the latter in a 0.5 per cent aqueous solution. Antiseptic collyria (usually zinc sulfate 0.03 Gm., boric acid 0.6 Gm. and distilled water to make 30 cc.) were generally used.

Insufficient treatment is an error often committed. Ten patients have been seen in whom dendritic lesions have been iodized by different members of the staff of the infirmary. In each of these cases the infective agent was recovered from the cornea a few days after treatment. This happens rarely if a proper application has been made. In this series of cases, after treatment a recurrence of corneal staining with fluorescein has been seen in many cases, but in only 2 has it been possible to prove the presence of the virus of herpes. In 1 of these cases iodine was applied only to the dendritic lesion, after which a new dendritic figure appeared on an entirely different area of the cornea a few days later. It is obvious that this type of recurrence has little to do with the treatment of the first lesion and is really analogous to the outbreak of a new fever sore on the other side of the mouth. In the other case a total application of iodine had been made to the cornea. In spite of this, a perfectly typical dendritic figure became apparent in the new epithelium three days later. Again it seemed probable that this represented a second "crop of lesions."

RESULTS OF TREATMENT WITH IODINE

Evaluation of treatment is exceedingly difficult when one is dealing with a disease which has so many variations. In no two cases is the disease exactly similar. The dimensions and configuration of the ulcer are never the same. Conjunctival congestion, hypesthesia, the amount of cellular infiltration surrounding the ulcer, the accompanying iritis and the old corneal scarring always vary. The preexisting duration of the disease and the patient's natural and acquired resistance, as well as the patient's age itself, probably play varying rôles. It is known that there are variations even in the virus of herpes itself. Dermotropic and neurotropic strains have been distinguished. The fact that the neurotropic strain may cause dendritic keratitis is suggested by the occasional death of a rabbit from herpes encephalitis after the cornea has been infected with material from the cornea of a human being with dendritic keratitis. For all these reasons it is practically impossible to use any of the foregoing criteria as a basis of classification. The alternative method of study has been chosen, i. e., that of combining all cases in which similar treatment was employed and charting the duration of corneal staining with fluorescein and the duration of conjunctival congestion after the tincture of iodine had been applied. The results in three groups of cases are presented.

CONTROL SERIES

The first group is a control series of 53 cases in which strong solution of iodine was not used. Generally speaking, these cases were of the milder nonprogressive type in which it was believed that more conservative treatment with various mydriatics and antiseptics was justified. Other patients were seen first in the subsiding phase of the disease, scrapings from their corneas often eliciting negative reactions on inoculation of rabbits, and for these patients it was felt that radical treatment was definitely contraindicated. The average duration of conjunctival congestion was forty-one days. The shortest period during which any one cornea stained was ten days, and the longest, forty-three. This last figure was obtained in a patient who exemplifies the most frequent complication of dendritic keratitis. Twenty

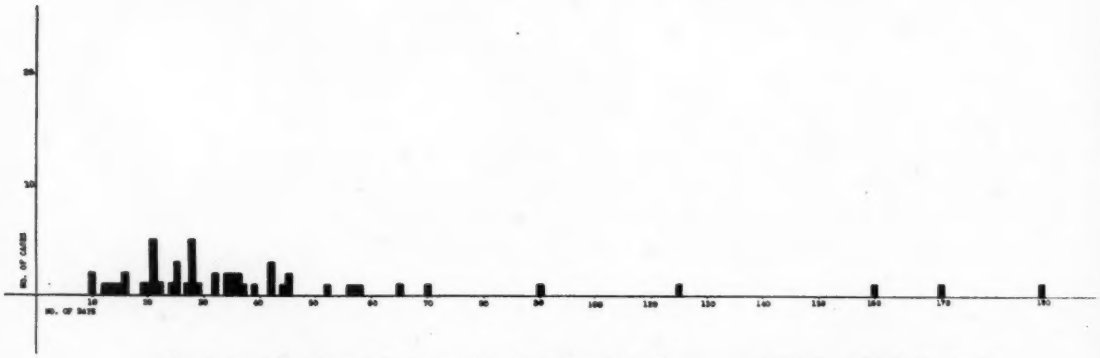

Chart 4.—Duration of congestion (in days) in the cases (53) in which tincture of iodine was not applied.

days after the dendritic ulcer had ceased staining, an attack of typical keratitis metaherpetica developed, and the cornea again showed a small ulcer, which continued to stain for fifty-five days. Repeated tests on rabbits were negative during this phase, as has invariably proved to be the case in a great number of tests made in cases of this type of recurrence. In this group of cases there were 11 which might be classified as complicated. Typical recurrences of keratitis metaherpetica developed in 9 of these. In 2 of the 9 the condition eventually developed into keratitis fascicularis and will be reported on at a later date. There was 1 case in this group of 11 in which disciform keratitis developed spontaneously, and another in which there was an accompanying hypopyon. If these complicated cases are entirely omitted from this grouping, the average period of staining (16 attacks) is twenty-one days and the average congestion (44 attacks) thirty-one days.

TREATMENT WITH STRONG SOLUTION OF IODINE LOCALLY

Cases in which strong solution of iodine was applied to the ulcer and the immediately surrounding area comprise the largest group. This type of treatment was used in 97 attacks in 87 patients. In general, the attacks were of more varied severity than those in the other groups, although in this group as a whole there were not as many severe attacks. The latter were more apt to be included in the group in which the entire cornea was treated with iodine. The average duration of corneal staining in all cases in which iodine was administered locally was seven and six-tenths days. The average duration of conjunctival congestion was fifteen and two-tenths days. It is of great therapeutic significance that in 82 of 97 attacks, or 85 per cent, there was no corneal staining ten

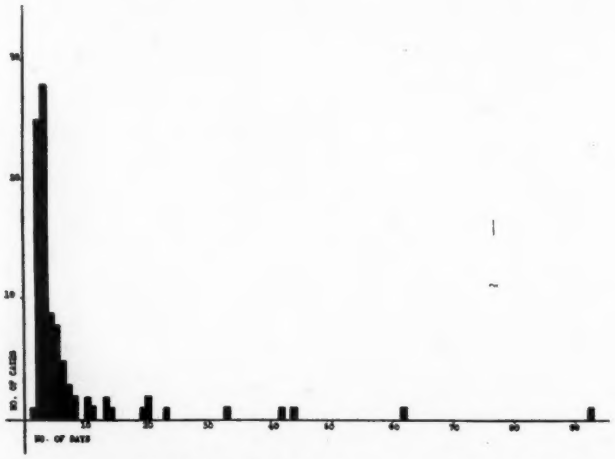

Chart 5.—Duration of staining (in days) in the cases (96) in which tincture of iodine was applied to the lesion only.

days after treatment, and in 70, or 72 per cent, no corneal staining after the fifth day. The duration of corneal staining and conjunctival congestion will be somewhat shortened if the following cases are excluded from this group: (1) 23 cases in which keratitis metaherpetica developed; (2) 2 cases in which hypopyon developed, and (3) 2 cases in which disciform keratitis developed. The average duration of corneal staining then becomes five and two-tenths days, and that of conjunctival congestion, ten and one-tenth days.

TREATMENT WITH STRONG SOLUTION OF IODINE TO ENTIRE CORNEA

The third group included those cases in which strong solution of iodine was applied over the entire cornea (45 attacks in 43 patients). It seems justifiable to subdivide this group because of the unusual severity of a great many of the attacks included. Two subgroups were

made arbitrarily on the basis of the clinical severity of the infection. Subgroup 1 comprised all cases in which the ulcer measured less than 12 mm. in length and 3 mm. in breadth, in which the conjunctival congestion was less than 4 plus on an arbitrary scale the highest reading

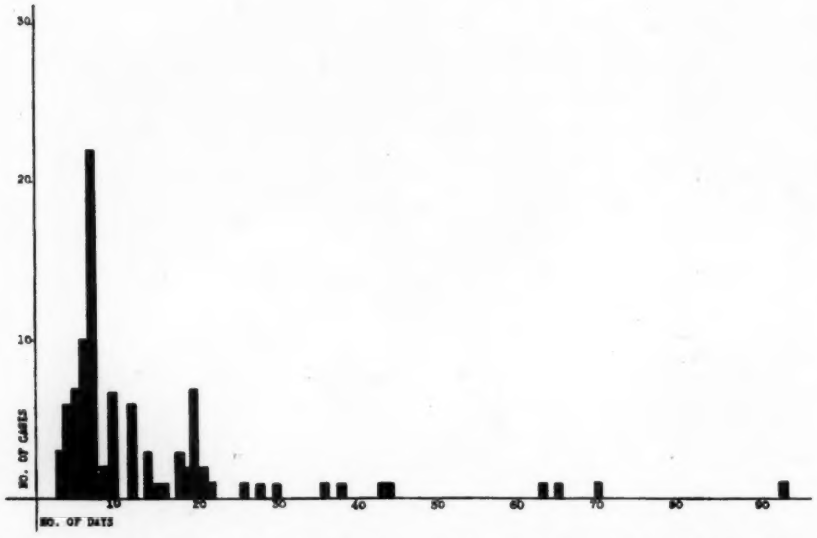

Chart 6.—Duration of congestion (in days) in the cases (97) in which tincture of iodine was applied to the lesion only.

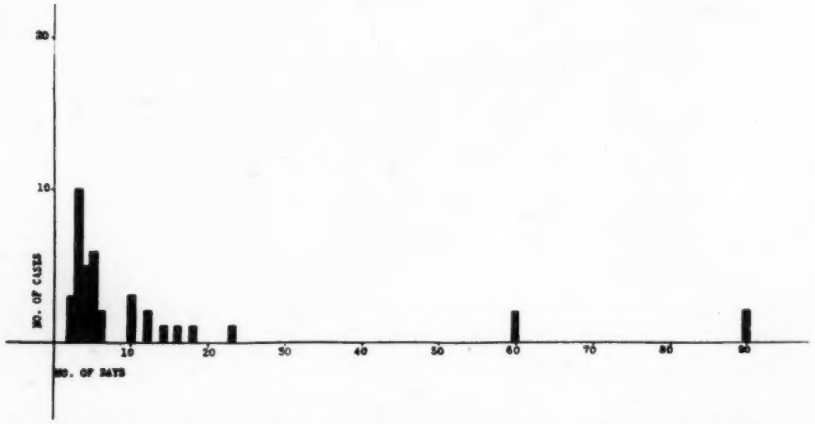

Chart 7.—Duration of staining (in days) in the cases (39) in which tincture of iodine was applied to the whole cornea.

of which was 4 plus and in which the secondary iritis was not attended by more than a few (from 10 to 20) cells in the aqueous per slit-lamp beam. Subgroup 2 comprised all cases the severity of which was greater than indicated by these specifications.

There were 19 cases included in subgroup 1 (milder cases). The average duration of corneal staining in these cases after application of iodine to the entire cornea was four and two-tenths days. The average duration of congestion was fourteen days. It is obvious from a comparison of these statistics with those from the group in which strong solution of iodine was applied to the dendritic ulcer alone that the amount of iodine does not materially influence the duration of corneal staining. Keratitis metaherpetica developed in 5 cases in subgroup 1. The average duration of corneal staining in this phase of the disease was thirty-three days. The shortest duration in a single case was two days; the longest was ninety days. There were 26 cases included in subgroup 2 (severe cases). The average duration of corneal staining after the application of iodine to the entire cornea was twenty days. The average dura-

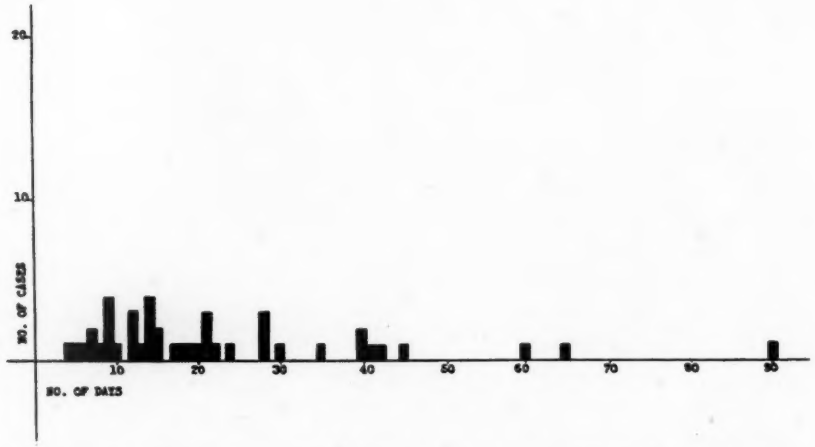

Chart 8.—Duration of congestion (in days) in the cases (43) in which tincture of iodine was applied to the entire cornea.

tion of congestion was twenty-eight days. Keratitis metaherpetica developed in 10 cases in this group. The average duration of corneal staining in this phase of the disease was thirty-five days. The shortest duration in a single case was seven days; the longest, ninety-six days.

BILATERAL DENDRITIC KERATITIS

The occurrence of dendritic keratitis in both corneas, either at the same time or at different times, is rare. In only 1 patient (F. P.) has the simultaneous bilateral occurrence been recognized.

This 58 year old man gave a history of having had poor vision in his right eye ever since he could remember, although he could not recall any previous attack of ocular inflammation. Thirty days before he was seen in the clinic, severe infection of the upper respiratory tract developed, with herpes simplex scattered

about the nose and mouth. Both eyes became sore and inflamed simultaneously. On examination there was marked congestion of the conjunctivae, and the corneas showed large staining dendritic ulcers. There was much more corneal infiltration in the right than in the left eye. Only in the left eye could the aqueous be clearly seen, and it contained a moderate number of cellular elements. Vision was reduced to 1/200 in the right eye and 20/200 in the left eye. Inoculation of rabbits was positive from the patient's left cornea only. Both ulcers healed promptly after one thorough application of iodine to each cornea on successive days.

Another patient (H. S.) had been seen in the clinic with four attacks of dendritic keratitis of his right eye. During the time of this study he appeared with typical dendritic keratitis of his left eye.

Five other patients with dendritic keratitis showed corneal scars in the noninflamed eye. Of these patients, one (M. M.) had been treated for bilateral interstitial keratitis in the clinic. In 2 patients (F. S. and N. S.) the scars were presumably due to tuberculosis. Both showed allergic reactions to skin tests with tuberculin in very high dilution and also showed signs of having had tuberculosis in childhood. The remaining 2 patients (M. B. and A. C.) showed corneal scarring which gave no clue to its origin. It is possible that the old keratitis may have been dendritic in nature. In summary, it may be said that one patient was seen with simultaneous bilateral dendritic keratitis, a second with dendritic keratitis of both eyes at different times and 5 with scars of the noninflamed cornea, some of which may have been from previous attacks of dendritic keratitis. In any event, dendritic keratitis of both corneas at the same time or at different times is exceedingly unusual. This fact is a very important point in the differential diagnosis of the disease.

RECURRENCE OF DENDRITIC KERATITIS AS SUCH

The frequency of recurrence of dendritic keratitis (not including cases of metaherpes) is of great interest. The 221 patients comprising this series were seen in 250 attacks. From their histories this same group has experienced a total of 466 attacks of dendritic keratitis, an average of 2.1 attacks per person. Eighty-nine patients were seen in their first attack, and 132 were seen who gave histories of previous similar episodes. These recurrences were all in the same eye except in 1 patient (H. S.), whose case was described previously. It is fair to say, therefore, that a single attack of dendritic keratitis produces a locus minoris resistentiae of high degree which makes this person greatly more subject to an attack than one who never has had the disease.

KERATITIS METAHERPETICA

The fact that corneal ulceration can recur in another form, that of keratitis metaherpetica, after the primary healing of a dendritic ulcer is shown clearly by the following 43 cases. It has been found that this phase of herpetic keratitis always occurs without demonstrable virus

in the ulcer, corneal epithelium or conjunctival sac. Fifty-seven inoculations of rabbits with material curetted from 29 of these ulcers gave negative results. Furthermore, material curetted from the surrounding corneal epithelium and from the conjunctival fornices has likewise produced no herpetic lesions in rabbits' corneas. Morphologically, keratitis metaherpetica is quite unlike dendritic keratitis, and the two diseases can practically always be differentiated clinically. The configuration of the ulcer is most important. In the metaherpetic phase one never finds the primary figures of dendritic keratitis often described as rosettes, Roman numerals or dendrites. The metaherpetic ulcers are small, usually round or somewhat oval, and very rarely show sinuous or scalloped borders as do the lesions in advanced severe dendritic keratitis. The epithelium also shows normal attachment to Bowman's mebrane instead of being very loosely attached, as it is in active dendritic

TABLE 3.—*Recurrences in the Control Series in the Form of Keratitis Metaherpetica After Spontaneous Healing (Nine Cases)*

Name	Age	Interval Before Recurrence, Days	Severity of Attack: Area of Ulcer, Mm.	Severity of Attack: Congestion	Duration of Recurrence, Days	
Adams	28	7	11 by 1	+++	?	(disciform keratitis developed)
Blake	77	17	7 by 1	+	90	
Leonard	53	60	10 by 2	+++	22	
Lydon	9	10	8 by 1	++	7	
Matulis	47	32	3 by 1	+	6	
Van Buskirk	19	20	9 by 1	+++	55	(fascicular keratitis developed)
McHugh	37	14	8 by 2	+++	60	(fascicular keratitis developed)
Puccia	72	10	9 by 2	++	150	(fascicular keratitis developed)
Rowen	8	15	12 by 1	+++	74	

Total number of cases in series, 53; recurrence, 17 per cent

keratitis. Multiplicity of minute ulcers is unusual but has been seen several times following dendritic keratitis in cases in which there have been several separate dendritic figures. Hypesthesia is pronounced and without doubt has given rise to the commonly used though less descriptive term "neuropathic" keratitis for the condition in these cases. Conjunctival congestion is minimal. It is not uncommon to make the diagnosis of keratitis metaherpetica in the case of an eye which is completely without congestion and which causes the patient little discomfort except slight tearing and possibly the sensation such as that produced by a foreign body.

The important details of the 43 cases of keratitis metaherpetica are given in tables 3, 4 and 5. Its incidence relative to the type of treatment used is as follows: 1. In the control series of 53 attacks in which iodine was not used, metaherpetic keratitis developed in 9 cases or 17 per cent. 2. Nineteen, or 20 per cent, of the 97 attacks of dendritic keratitis in which iodine was applied to the ulcer alone were followed by this type of recurrence. 3. In 15, or 33 per cent, of the 45 attacks in which iodization

of the entire cornea was used there were recurrences in this form. As might be expected, the highest incidence occurred with the most intensive use of iodine. However, this treatment cannot be accepted as the chief

TABLE 4.—*Recurrences in the Form of Keratitis Metaherpetica After Application of Iodine to Dendritic Lesion Only (Nineteen Cases)*

Name	Age	Duration Before Iodine, Days	Interval Before Recurrence, Days	Severity of Attack		Duration of Recurrence, Days
				Area of Ulcer, Mm.	Congestion	
Brady	6	2	4	10 by 2	++	22
Browning	10	8	12	15 by 1	++	?
Carr	66	9	70	11 by 1	+	56
Connell	71	15	14	10 by 8	+++	7
Greer	6	14	21	6 by 1	++	27
Knox	17	9	9	10 by 2	++	26
McGinn	22	17	7	6 by 4	+++	3
McLaughlin	52	34	8	8 by 3	+++	?
Morris	14	7	7	23 by 3	+++	23
Sawyer	34	10	11	11 by 1	+++	10
Sherman	48	9	4	9 by 3	+++	65(intermittent staining)
Thomas	60	2	5	8 by 1	++	2
Connolley	35	11	2	4 by 1	+++	15
Hogan	54	7	6	6 by 1	++	20
Herrick	47	21	7	4 by 1	++	20
Johnston	31	57	7	10 by 3	+++	4
Pisani	10	15	7	9 by 1	++	30
Rive	42	6	14	6 by 2	+++	150
Tulip	13	6	11	8 by 1	++	5

Total number of cases in which iodine was applied to lesion only, 97; recurrence, 19.6 per cent

TABLE 5.—*Recurrences in the Form of Keratitis Metaherpetica After Application of Iodine to Entire Cornea (Fifteen Cases)*

Name	Age	Duration Before Iodine, Days	Interval Before Recurrence, Days	Severity of Attack		Duration of Recurrence, Days
				Area of Ulcer, Mm.	Congestion	
Anello	12	8	10	11 by 1	+	14
Bailey	15	28	14	14 by 1	++	56
Baue	53	11	17	10 by 2	+++	90
Castignatti	12	15	10	8 by 2	+++	21
Fessenden	55	75	5	26 by 2	+++	14
Flaherty	54	73	21	10 by 6	++++	14
Ford	21	2	30	10 by 1	+++	2
Gorman	47	15	7	6 by 3	+++	51
Kelly	50	7	28	15 by 3	+++	21
Luzio	54	18	25	14 by 3	+++	57
Marshall	13	4	2	18 by 3	++	7
Parker	40	21	2	10 by 3	+++	96
Serol	40	28	11	9 by 1	+++	44
White	50	4	20	8 by 1	++	7
Nixon	64	38	7	12 by 1	+++	?

Total number of cases in which iodine was applied to entire cornea, 45; recurrence, 33 per cent

factor in the cause of these recurrences, since the disease recurred in this form in 17 per cent of the cases in which iodine was not used. The incidence of metaherpetic keratitis shows no constant relationship to the size of the original ulcer, amount of congestion, severity of accompanying iritis or the duration of the disease before treatment was instituted. Neither does the disease show a marked preference for any age group.

OCULAR HERPES WITHOUT CORNEAL INVOLVEMENT

Five patients showed recurrence of ocular herpes without corneal involvement (table 6). Each had been seen previously with one or more attacks of ipsilateral dendritic keratitis. Congestion of the conjunctiva with herpes febrilis of the lids occurred in cases 1, 2 and 5. Congestion of the conjunctiva alone appeared in only 1 case (case 3). In none of these 4 patients could any vesicular lesions be found in the conjunctiva.

The fact that iritis often accompanies dendritic keratitis is well known, but in every case it is probable that toxic products from the cornea alone are capable of producing irritation of the iris. Inoculations of the corneas of 2 rabbits with the aqueous from 2 patients so affected have not demonstrated the presence of virus in the anterior chamber. Case 4 is of significance in this connection because the patient previously had 2 attacks of dendritic keratitis in this eye. About two

TABLE 6.—*Recurrence of Ocular Herpes Without Corneal Involvement (Five Cases)*

Name	Age	Type of Recurrence	Original Attack
Brady.....	8	Congestion of conjunctiva with herpes of lids	Dendritic keratitis; 3 previous attacks
Bullock....	19	Congestion of conjunctiva with herpes of lips	Dendritic keratitis; very severe attack 18 months before
Byam.....	23	Congestion of conjunctiva alone.....	Dendritic keratitis; 4 previous attacks
Leonard...	53	Severe iritis alone.....	Dendritic keratitis; 2 previous attacks
Levin.....	7	Congestion of conjunctiva with herpes of lids	Dendritic keratitis; 2 previous attacks

years after the second attack of dendritic keratitis acute iritis developed in the same eye. The iritis was characterized by little pain, moderate congestion, many engorged vessels in the iris, and many cellular elements in the aqueous. There was some detritus on the posterior corneal surface but no visible fibrin in the anterior chamber. The attack subsided completely in fourteen days with the use of atropine. No inoculation was made from the aqueous in this case, since secondary glaucoma was not present and paracentesis was not justified, but the previous occurrences of dendritic keratitis strongly indicate that the iritis was due to the virus of herpes.

DENDRITIC KERATITIS ASSOCIATED WITH HYPOPYON

Table 7 shows that dendritic keratitis not uncommonly is the underlying cause of hypopyon ulcers. Thirteen cases are reported. In 4 of these the condition was proved to be herpetic by positive inoculation of rabbits; the remaining 9 were suspected of being cases of dendritic keratitis because of previous attacks of this condition, the configura-

tion of the corneal lesions and the absence of pathogenic organisms in smears and cultures of material taken from the ulcers. These ulcers were rarely as destructive as true ulcus serpens and occasionally healed without electrocauterization or iodization, although these more radical forms of treatment were very effective in the majority of the cases.

DISCIFORM KERATITIS OF HERPETIC ORIGIN

The virus of herpes has been recovered from the corneas of 3 patients with disciform keratitis during the past forty-two months. Two of these patients when first seen had dendritic keratitis which changed its character very quickly without treatment and became indistinguishable from true disciform keratitis. The third patient from whose cornea virus was recovered was seen only after the disciform lesion was well developed. In a fourth patient, who had an attack of unmistakable dendritic keratitis, a severe disciform keratitis developed after application of iodine to the ulcer only. No rabbit test was made in this instance. It may be of some importance that this patient had acquired syphilis also. These cases will be reported in detail elsewhere.

EXPERIENCES WITH OTHER FORMS OF TREATMENT FOR DENDRITIC KERATITIS

Alcohol.—In an attempt to determine whether the constituent parts of strong solution of iodine were as effective as the solution itself, alcohol was applied to 2 dendritic ulcers. Applications were made in the same manner as were those in which strong solution of iodine had been used. The first patient (H. M.) treated in this manner had a severe proved attack of dendritic keratitis associated with pulmonary tuberculosis. Absolute alcohol was applied to the entire ulcer. Although some improvement was noticed in four days, the ulcer looked active, and strong solution of iodine was then applied. Four days following this application of iodine the cornea showed no staining. The second patient (E. S.) had a mild attack of dendritic keratitis of ten days' duration. Alcohol (90 per cent) was applied to the ulcer. Corneal staining ceased seven days thereafter, and the eye was white and quiet on the tenth day. Judging from these two experiences only, it seems that alcohol might be effective if its dosage could be controlled more accurately. The objection to the use of alcohol is that it does not stain the cornea, and therefore the amount of treatment cannot be controlled.

Potassium Iodide.—A 5 per cent solution of potassium iodide was instilled every two hours for one week into the conjunctival sac of a patient (H. L.) with dendritic keratitis. No change could be noticed in the appearance of the ulcer. It was concluded that the treatment was not effective.

TABLE 7.—*Dendritic Keratitis Associated with Hypopyon (Thirteen Cases)*

Name	Age	History	Examination	Bacteriology	Rabbit Test	Comments
Cozzaglio.....	50	Duration, 70 days	Typical dendritic keratitis	Not done	Not done	Prompt healing after actual cautery, application of iodine to entire cornea, and conjunctival flap; duration 3 weeks after operation
Elliot.....	47	Duration, 7 days; acquired syphilis	Typical dendritic keratitis	Smear and culture negative	Not done	Disciform keratitis developed after application of iodine to lesion only; 2 mm. hypopyon in fourth month; duration 20 weeks
Haggie.....	16	Dendritic keratitis 13 years previously; duration, 14 days	Severe dendritic keratitis	Xerosis and rare Staphylococcus albus	Positive 14th day	2 mm. hypopyon developed 5 days after application of iodine to entire cornea; duration 10 weeks
Harrington.....	52	No previous attacks; duration, 60 days	Clinically, dendritic keratitis with 4 mm. hypopyon	Not done	Not done	Healed after electrocauterization and conjunctival flap; duration 4 weeks after operation
Kearns.....	36	No previous attacks; duration, 65 days	Clinically, dendritic keratitis with 4 mm. hypopyon	Not done	Positive 65th day	Healed after thermophore; duration 6 weeks after operation
Mahoney.....	52	Eight previous attacks; duration, 18 days	Clinically, suggestive of dendritic keratitis with 2 mm. hypopyon	Diphtheroids	Negative 15th and 17th day	Application of iodine not effective; healed after electrocauterization and flap; duration 3 weeks after cauterization
Movello.....	53	No previous attacks; struck by stone at onset	Clinically, dendritic keratitis	Not done	Negative 13th day	Developed 2 mm. hypopyon 3 days after application of iodine to ulcer only; healed after electrocauterization; duration 6 weeks after cauterization
Murphy, M.....	74	Two previous attacks of hypopyon ulcers	Typical dendritic keratitis; no hypopyon with this attack	Not done	Positive 4th day	Healed 3 weeks after application of iodine to ulcer only
Murphy, R.....	6	Hypopyon ulcer 3 years ago	Typical dendritic keratitis; no hypopyon with this attack	Not done	Positive 21st day	Healed 2 weeks after application of iodine to ulcer only
Quain.....	55	Severe cold with herpes labialis at onset; duration, 45 days	Clinically, dendritic keratitis with 3 mm. hypopyon	Smear and culture negative	Negative 30th and 45th day	Healed 3 weeks after application of iodine to ulcer only
Russell.....	61	Two previous attacks; duration, 10 days	Clinically, dendritic keratitis with 1 mm. hypopyon	Smear negative; culture, few colonies of staphylococci	Negative 10th day	Healed without cauterization; duration 5 weeks
Tobey.....	64	No previous attacks; duration, 35 days	Clinically suggestive of dendritic keratitis with 2 mm. hypopyon	Not done	Negative 35th day	Healed after local applications of heat, atropine and antiseptics; duration 4 weeks
Whitcomb.....	65	No previous attacks; duration, 60 days	Clinically, dendritic keratitis with 2 mm. hypopyon	Smear negative; culture xerosis	Negative 60th day	Healed after application of iodine to entire cornea

Hydrogen Peroxide.—It is well known that the virus of herpes is very easily destroyed by oxidizing agents. Therefore, it was thought that careful application of neutral hydrogen peroxide to the ulcer itself might be very effective in the treatment of dendritic keratitis. Two patients (A. P. and J. D.) with moderately severe attacks, proved by inoculation of a rabbit, were thus treated. No curative effect was noted in either case. In this connection it should also be noted that although hydrogen peroxide acts as a very mild antiseptic when used in the mouth and open wounds, it is very painful and destructive to corneal tissue. A single instillation into the conjunctival sac of a rabbit caused a complete and permanent opacity of the cornea.

Epinephrine.—The application of epinephrine hydrochloride (1:1,000) to the cornea under a contact glass combines two possible methods of destroying the virus of herpes in cases of dendritic keratitis. Epinephrine is a very strong reducing agent, and the ordinary preparations of it are acid in reaction (p_H, approximately 5.6). The effect of the acidity was of chief interest. Three patients suitable for this experiment were chosen. The first (J. R.) wore a contact glass filled with epinephrine hydrochloride for fifteen minutes. Six days later, as the ulcer had shown no change and a positive reaction was obtained on inoculation of a rabbit, strong solution of iodine was applied, after which the ulcer healed promptly. The result obtained in the second case (W. S.) was very similar to that of the first. Epinephrine hydrochloride was applied for fifteen minutes on the thirtieth day of the disease. Three days later there was no improvement, and inoculation of a rabbit gave positive results. Iodization was resorted to in this case also, and there was no staining of the cornea two days thereafter. Scrapings from the ulcer in the third case (J. V.) elicited a positive reaction on inoculation of a rabbit on the twelfth day of the disease. A contact glass full of epinephrine hydrochloride was placed over the cornea for twenty minutes immediately after curetting the ulcer for inoculation. There was no perceptible improvement in this case. Staining continued for thirty days, although inoculation of a rabbit seven days after the treatment gave negative results.

Curettage.—The beneficial effect of curettage alone is well demonstrated by the following case (F. D.): The patient had a small primary figure on the cornea. Inoculation of a rabbit on the ninth day of the disease gave negative results. Since there was no significant change in the cornea two days later and because of a strong desire to demonstrate virus in this typical case, the entire epithelium was removed in the affected area, and inoculation of a rabbit with this material gave positive results. The cornea of the patient then showed signs of improvement, and staining ceased on the thirteenth postoperative day.

Alkalis.—The effect of local application of alkalis to the cornea has not been tried, but with the use of sodium bicarbonate as internal medication for herpes febrilis in mind, this drug was prescribed in large doses for 3 patients (J. C., R. F. and J. L.) with dendritic keratitis. No curative effect could be observed.

Vaccinations.—Two types of vaccination were attempted. Following the idea that a higher immunity might develop if herpes simplex could be artificially produced on some other part of the body, the lips of 4 patients (G. G., J. K., T. H. and J. M.) were vaccinated with virus from their own dendritic ulcers. No vesicles developed, and no improvement was observed in their corneal ulcers.

The second type of vaccination made was with cowpox vaccine. These vaccinations were performed in 8 cases in the usual manner on the patient's arm or leg. It was hoped that immunity for vaccinia might be effective for the closely related disease herpes. In none of the 8 cases, however, was there any observable effect on the keratitis. Moreover, in 4 cases that were followed there were recurrences within two years. These results indicate that immunity to vaccinia neither cures the lesions of herpes nor prevents their recurrence.

Paracentesis of the Anterior Chamber.—One patient (A. Q.) had an unusually severe attack of dendritic keratitis of thirty days' duration. No improvement was noticed after a week of treatment with conservative measures; in fact, a small hypopyon had developed. Paracenteses of the anterior chamber were performed on the thirty-seventh and thirty-ninth days in the hope that a change of aqueous might effect a cure. The patient's cornea showed no improvement until after an application of iodine was made on the forty-fifth day.

Ultraviolet Irradiation.—The germicidal effect of ultraviolet rays on the virus of herpes has been previously investigated by me.[3] It was concluded that the virus of herpes could not be destroyed in the cornea by any exposure to ultraviolet rays which would be practical to use on the human eye. Furthermore, it was concluded that if the beneficial clinical results reported in cases in human beings were due to ultraviolet irradiation, they could not be attributed to the germicidal effect of the rays but must be due to some unknown secondary effects. An explanation for a secondary effect is indicated by the following 2 cases: P. C. was seen on the twelfth day of an attack of dendritic keratitis with a small primary figure on the cornea. A minimal erythema dose of ultraviolet radiation was applied to the entire cornea by means of a water-cooled quartz mercury vapor arc lamp. A rabbit was inoculated directly after the treatment, with positive results. Six hours after the

3. Gundersen, T.: Germicidal Effect of Ultra-Violet Rays on the Virus of Herpes, Arch. Ophth. **8**:519 (Oct.) 1932.

treatment, severe ocular pain developed, which persisted for about ten hours. The day following irradiation practically two thirds of the corneal epithelium was found denuded. In spite of the positive reaction on inoculation of a rabbit directly after irradiation, the cornea showed prompt healing. Corneal staining ceased six days after treatment, and the conjunctival congestion disappeared on the eighth day. C. F. was seen first on the seventh day of an attack of typical primary dendritic keratitis, slightly more severe than that in the first case. Inoculation of a rabbit gave positive results. Three days later a suberythema dose of ultraviolet rays was applied to the patient's cornea. This was also followed by symptoms of electric ophthalmia, although they were not as severe as in the first case. Twenty-four hours after irradiation there was increased conjunctival congestion, but no loss of corneal epithelium. This patient was under careful observation for eleven days after irradiation but showed no evidence of a curative effect from the treatment. Tincture of iodine was therefore applied to the entire corneal surface, and healing of the dendritic ulcer promptly followed. Corneal staining disappeared after three days, and conjunctival congestion, after twelve days.

It is apparent from the foregoing results that whether treatment is by irradiation, by alcohol or by curettage the corneal epithelium surrounding the entire ulcer must be destroyed in order to effect a cure. In cases in which this has not been accomplished, there are no obvious beneficial effects. This is in accord with the modern conception of virus diseases in general, which is that although the infective agent of itself is very labile, once the virus has invaded tissue cells it is impervious to antiseptics which do not destroy the cells themselves. The satisfactory results in the treatment of dendritic keratitis with strong solution of iodine can be attributed primarily to its destructive effect on the corneal epithelium without penetration of Bowman's membrane.

SUMMARY AND CONCLUSIONS

A study is reported of 250 attacks of dendritic keratitis in 225 patients.

Among the patients admitted to the outpatient department of the Massachusetts Eye and Ear Infirmary during the past forty-two months, dendritic keratitis was the most frequent corneal infection recognizable on an etiologic or morphologic basis.

A total of 623 inoculations of rabbits was made with scrapings from the corneas of patients with various types of corneal disease. The material for 180 of these inoculations was obtained from patients whose condition was diagnosed clinically as dendritic keratitis. Sixty-three per cent of the 180 inoculations gave positive results. Later, in cases

in which the clinical picture was perfectly typical, tests were not made on rabbits. The percentage of positive reactions would doubtless have been somewhat higher had the tests been made in the latter group of cases.

In no type of corneal disease other than dendritic keratitis and disciform keratitis has the virus of herpes simplex been demonstrated.

Dendritic keratitis appeared about two and a half times as frequently in adult males as in adult females, although in patients under 16 years of age the sex incidence was equal.

The incidence of dendritic keratitis was found to be highest before the tenth year of life.

There was no striking seasonal incidence, although the lowest number of attacks originated during September and October.

Herpes febrilis appeared elsewhere on the face 76 times in 203 attacks, or in 37.4 per cent, in which this observation was recorded.

Colds, grip and other infections of the upper respiratory tract were seen associated with dendritic keratitis in 118 of 190 attacks, or in 62.1 per cent.

The data from these cases considered from various points of view afford no conclusive evidence that trauma is a contributory cause of dendritic keratitis.

Evidence from the clinical study of these cases indicates that corneal hypesthesia follows rather than precedes attacks of dendritic keratitis.

Strong solution of iodine has been applied to the dendritic ulcer and immediately surrounding area of the cornea in 97 attacks and to the entire cornea in 45 attacks. The results of this treatment have been compared with those in a control series of 55 cases.

It has been observed that the application of strong solution of iodine has not contributed to the amount of corneal scarring in any uncomplicated case.

A case of simultaneous bilateral dendritic keratitis is reported; another case is reported in which the disease occurred in both eyes at different times.

Three types of recurrence of ocular herpes were observed, namely, dendritic keratitis as such, keratitis metaherpetica and ocular herpes without corneal involvement.

Thirteen cases of dendritic ulcer associated with hypopyon were seen. The virus of herpes was demonstrated in 4 of these.

A preliminary report is made of 4 cases of disciform keratitis of herpetic origin. The virus of herpes was demonstrated in 3.

Experiences with the following other therapeutic agents are reported: alcohol, potassium iodide, hydrogen peroxide, epinephrine hydrochloride under a contact glass, curettage, alkalis, vaccination, paracentesis of the anterior chamber and ultraviolet radiation.

It is concluded that application of strong solution of iodine is the most effective treatment for dendritic keratitis known at present. It is believed that its effectiveness depends chiefly, if not entirely, on the destruction of the infected corneal epithelium and that no other form of treatment can be successful unless it destroys the tissues which harbor the virus.

ABSTRACT OF DISCUSSION

Dr. Everett L. Goar, Houston, Texas: I have seen comparatively few children with dendritic keratitis. Most of the children that I have seen with keratitis had a different type of lesion, usually a phlyctenular, interstitial or marginal ulcer of undetermined origin. I doubt whether trauma plays any active part in this condition, although it undoubtedly occurs fairly frequently in corneas that have been vascularized from previous infections.

While malaria was formerly considered an etiologic factor in dendritic keratitis, this can now definitely be ruled out in the majority of cases. Several years ago the blood of a series of patients was examined, and in no case could the plasmodium be found. Not infrequently a patient with dendritic keratitis gives a history of some febrile disorder of short duration just previous to the appearance of the corneal lesion. Such persons often have labial or palpebral herpes at the same time. It is surprising that the author did not encounter a patient with vesicular herpetic keratitis; in fact, from the title of the paper one would suppose that dendritic keratitis is the only type of herpes that affects the cornea. This is not in accordance with my experience. It is true that one does not see the vesicular type before the tiny vesicles have ruptured, but the discrete, tiny areas that appear suddenly stud the superficial layer of the cornea and disappear without coalescing are seen frequently in my practice.

While this condition is much more evanescent than the dendritic type, it is just as surely a herpetic keratitis. I have always regarded the lesions that appear during or after an attack of dendritic keratitis, which Dr. Gundersen calls keratitis metaherpetica. as a phase of the same disease with different configuration. Certainly the cornea about these lesions shows the same hypesthesia and the areas are likely to heal with as much difficulty as the original ulcer. In spite of the failure to produce them experimentally in the rabbit, I cannot conceive of this as a separate entity.

I have used strong solution of iodine locally, just as Dr. Gundersen described, in a great many eyes with dendritic keratitis. I abandoned it once in favor of thorough curettement, and I now use curettement followed by the application of strong solution of iodine. The result to be attained is the removal of the necrotic tissue and enough apparently healthy epithelium so that no virus remains to produce extension. This must be done without injury to Bowman's membrane. Any method that accomplishes this is effectual, but the iodine is particularly useful because it destroys the epithelial cells adjacent to the ulcer, and a timid curettement will not do this. I do not agree with Dr. Gundersen that the eye should be left open. Any condition in which a large area of

corneal epithelium is abraded does better under a bandage; in fact, it does better with the eyes immobilized as much as possible. In severe cases there is always some irititis, and I believe that mydriatics are useful.

Dr. Jonas S. Friedenwald, Baltimore: Modern knowledge of herpetic infections dates from the experiments of Grüter, who discovered in 1913 that the disease could be transmitted to rabbits. During the period of the World War little attention was paid to this discovery, but about 1919 a great many workers in many laboratories began to study the problem of herpetic infections, and from then until about 1925 or 1926 numerous important contributions to the knowledge of this subject were continually appearing. Though numerous studies have been made during the last decade, little has been added to the knowledge, and I think that it is a tribute to Dr. Gundersen's insight to reopen the field.

In regard to the efficacy of treatment with iodine, I am in thorough agreement and have used it for years. I believe with him that the chief cause of failure of this type of treatment is the inadequate use.

Dr. Gundersen suggests that the reason for the failure of iodine in experimental keratitis in rabbits is the absence of Bowman's membrane. That may perhaps be true, for the application of iodine to the eye of a rabbit produces a very dense corneal opacity. My experience in experimental herpetic keratitis has been that in addition to the opacity produced there is also failure of the iodine to stop the progress of the disease. Failure to obtain successful results in treatment of the experimental disease does not signify that the results on human beings are to be questioned, for the disease in rabbits is essentially different from that in human beings. In man it is a chronic disorder which does not lead, as a rule, to immunity. As Dr. Gundersen has shown, the incidence of recurrence is high. In rabbits, the disease is acute and leads to permanent and complete immunity.

One method of removing the corneal epithelium in a most satisfactory and complete manner without producing any injury to the corneal stroma is the application of bile. I think that it would be worth while to test the effect of bile as a possible therapeutic agent in these cases.

It would also be interesting to know how widely the virus is distributed in the bodies of these patients. In cases of herpes labialis the virus is not infrequently found in the spinal fluid.

Dr. Trygve Gundersen, Boston: In regard to the ordinary conception that herpes of the cornea occurs as an outburst of small fine vesicular lesions of the cornea, I repeat that I have never seen a herpetic vesicle occur on the cornea either in man or in an experimental animal. On the other hand, it is well known that in cases of bullous keratitis one may see vesicles in all stages of development, from those of fine pinpoint size to those measuring several millimeters in diameter. If vesicles do occur in cases of herpetic keratitis, why should they rupture before one sees them?

In regard to Dr. Friedenwald's suggestion, I must say that I am extremely interested in the application of bile to the cornea. I have not tried it. If bile will remove the corneal epithelium with less pain than iodine does, I think that it would be very valuable.

RÔLE OF MALARIA IN CONTROL OF ATROPHY OF THE OPTIC NERVE DUE TO SYPHILIS

A STUDY OF TWELVE CASES

C. P. CLARK, M.D.
INDIANAPOLIS

The fever treatment of syphilitic diseases of the central nervous system has commanded considerable space in the medical literature during the past decade. In 1918 Wagner von Jauregg [1] announced that he had intentionally inoculated patients suffering from dementia paralytica with the organism of tertian malaria and as a result of this experiment had witnessed physical and mental improvement in a certain percentage of the patients. Since then the treatment of patients suffering from dementia paralytica by induced malaria has been placed on a firm basis and is now recognized as a sound therapeutic procedure. Fever has also been induced in persons with other syphilitic conditions either by a disease produced intentionally or by some artificial method of elevating the body temperature. At the present time there is a wave of enthusiasm throughout the United States over the use of artificial or mechanical means of elevating the body temperature of the patient who is afflicted with syphilis, while the European physicians, especially the Germans, cling to the induction of malaria.

In 1888 Welch [2] advanced the theory that fever is one of the body's defenses against disease rather than a symptom of it. Since then this theory has gradually gained acceptance. Other physical phenomena were considered to be the efforts of the host to overcome invasion by the respective disease-producing organisms. Several procedures which have been known for a long time are used to stimulate the body defenses: the production of a sterile abscess by the injection of a sterile tissue irritant into the tissues, the injection of a protein substance into the muscle and the intravenous injection of a small quantity of a vaccine. A chill followed by fever is a reaction commonly witnessed after any of the foregoing procedures. It has been known for many years that the altered conditions incident to an acute febrile disease may be antagonistic to the continuation of a primary chronic illness. The patient recovers from both diseases as a result of the stimulation of the defense forces of the body by the acute febrile disease. Wagner von Jauregg

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From the Department of Ophthalmology, Indiana University School of Medicine, and the Indianapolis City Hospital.

1. von Jauregg, Wagner: Psychiat.-neurol. Wchnschr. **20**:132, 1918.

2. Welch, W. H.: The Cartwright Lectures on the General Pathology of Fever: Lectures II and III, M. Rec. **33**:401 (April 14); 457 (April 28) 1888.

made use of this knowledge when he began his important experiments in the treatment of patients with dementia paralytica. He knew that some of these patients who became infected with malaria in the natural manner recovered from the dementia paralytica. The use of induced malaria to treat patients afflicted with dementia paralytica and other forms of syphilis of the central nervous system began with this epoch-making work. Attention has been centered on the elevation of body temperature, with little regard shown for the other factors of body defense generated by the induced disease. The German physicians maintain that elevation of body temperature is but one factor in the defense offered the patient by malaria and that the important factor in the defense developed is stimulation of the reticulo-endothelial system.

In 1930 I [3] reported on the ocular conditions and changes observed in fifty patients suffering from dementia paralytica after treatment with tertian malaria. It was pointed out that the pupillary response to light stimuli could be recovered providing the treatment was instituted early. Also, visual fields were observed to be enlarged when the syphilitic destruction of the optic nerves and visual pathways was not complete and before diffuse gliosis developed.

In 1934 I [4] made another report concerning the effect of malaria on certain syphilitic conditions of the eye. The records of sixty patients were used in this work. These patients were suffering from various forms of syphilis of the central nervous system. All of them were inmates of a state hospital for patients with mental disease. It was concluded from this study that one could not expect marked improvement in the central visual acuity after treatment with malaria; that syphilitic invasion of the optic nerves may be arrested provided the treatment is instituted early, and that the Argyll Robertson pupillary phenomenon may disappear if treatment with malaria is instituted before permanent damage has occurred to the area or areas of the brain that control the reflexes of the pupils.

No inflammatory ocular conditions were investigated in either study. There was no recent paralysis of the ocular muscles in either group, and no changes in the muscles were observed after malaria was terminated. It must be remembered that inflammatory ocular conditions offer a better prognosis than disease of nerve tissue, especially nerve tissue that is associated with vision. In the former there may be, and many times there is, complete recovery of function when the ocular inflammation has subsided, while there can be much less hope for full recovery of function of the nerve tissue after the active disease process

3. Clark, C. P.: Eye Changes Observed in Paretic Patients After Treatment with Malaria, Am. J. Ophth. **13**:946, 1930.

4. Clark, C. P.: The Effect of Malaria upon Certain Syphilitic Conditions of the Eye, Tr. Am. Ophth. Soc. **32**:452, 1934.

has subsided. In the latter the best that one can expect is to arrest the destructive process, and one can anticipate permanent partial impairment. The treatment should be considered a therapeutic victory if complete loss of vision does not occur.

The following case histories are reported to show that malaria has the power of arresting the syphilitic invasion of the optic nerve and visual pathways when that invasion has not been of sufficient intensity to produce complete destruction, that blindness is inevitable when there is complete or diffuse syphilitic invasion of these tissues and that the examiner cannot accurately estimate before malaria treatment the amount of nerve destruction that will result from the syphilitic invasion of the tissues.

REPORT OF CASES

CASE 1 (Tabes).—E. A., a white man, aged 48, married, was admitted to the ophthalmologic clinic of the Indianapolis City Dispensary on Aug. 11, 1933, with the complaint of poor vision and inability to see toward the left. His wife had had no miscarriages or stillbirths. There were three healthy children, aged 16, 14 and 11 years, respectively. The patient said that he had never had venereal disease and had received no treatment for syphilis. He did not use alcohol or tobacco. One year previous to his admission to the clinic he noticed that his vision was blurred and consequently patronized an advertising optometrist in a department store. He was given glasses which were subsequently changed twice by the optometrist. However, the visual impairment increased, especially for newspaper print.

Central vision in the right eye was 15/200 and in the left eye, 20/200. The pupils were unequal in size, the left being larger than the right. The right pupil was round, and the left, oval. The pupils were immobile to light stimuli. Both responded well in convergence. Ocular motility was unimpaired. The ocular fundi presented very pale disks; the lamina cribrosa was visible over practically the entire surface. The arteries were constricted. The visual fields were as shown in chart 1. The knee jerks were absent, and the Romberg sign was present. No disturbance of speech was elicited. There was complement fixation of the blood; the Wassermann and Kline reactions were 4 plus. Examination of the spinal fluid disclosed a 4 plus Wassermann reaction and a colloidal gold curve of 4444433210.

The patient was referred to the hospital for malaria treatment. Some difficulty was experienced in finding a suitable donor. Two injections of typhoid vaccine were given intravenously, consisting of 25,000,000 and 50,000,000 organisms, respectively. This produced a temperature of 100 and 102 F., respectively.

On September 21, 10 cc. of blood from a patient with malaria was administered, and the first paroxysm of fever occurred on September 28. Thirteen paroxysms of fever with a temperature above 101 F. occurred before quinine was given to terminate the malaria.

The patient returned to the clinic on Oct. 13, 1934. The condition of the eye and vision were unchanged.

On May 4 central vision was 20/200 in the right eye and 10/200 in the left eye. The pupils and ocular fundi were unchanged. Examination of the spinal fluid disclosed a 4 plus Wassermann reaction and a colloidal gold curve of 0022211100.

On December 15 central vision was 20/100 in the right eye and 20/200 in the left eye. The pupils were approximately equal in size, round, one third dilated and immobile to light stimuli and responded well in convergence. The fundi showed advanced primary atrophy of the disks. The fields were as shown in chart 1.

CASE 2 (Tabes).—W. H., a Negro aged 35, was admitted to the ophthalmologic clinic of the Indianapolis City Dispensary on Aug. 4, 1933, with the complaint of poor vision. He had received antisyphilitic treatment at intervals during the past three years. The Wassermann and Kline reactions of the blood were 4 plus. The knee jerks were abolished, and the Romberg sign was present. Central vision in the right eye was limited to perception of light; in the left eye it was 5/200. The left pupil was larger than the right, and both were round; both reacted to direct and to consensual light stimuli. The convergence reflex was barely present. The right eye deviated lateralward, and a convergence effort could scarcely be obtained. The ocular motility was otherwise normal.

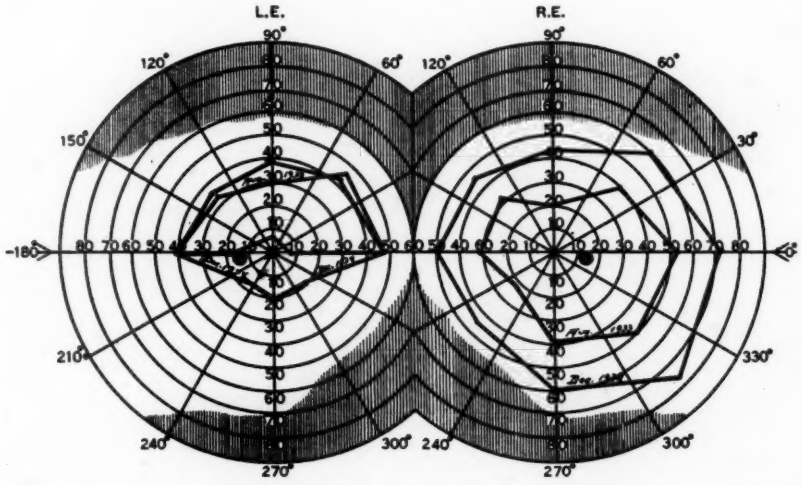

Chart 1 (case 1).—Visual fields as found in August 1933 and in December 1934. A 2 mm. test object was used at a radius of 330 mm.

There was marked primary atrophy of each optic disk. A small crescent of pale pink tissue was present at the upper nasal quadrant of the left disk. The arteries were threadlike in appearance. The field for the left eye was as shown in chart 2.

The patient was inoculated with the organism of tertian malaria. Six paroxysms of fever with a temperature above 101 F. occurred between September 28 and October 17, when quinine was administered. There was subjective visual improvement.

On May 6, 1934, central vision in the right eye was limited to perception of light; in the left eye it was 20/200. The pupils were round and equal in size; the right one reacted feebly to direct light and promptly to consensual light stimuli; the left one had a feeble response to consensual light stimuli but a good response to direct light stimuli. The right eye was divergent. There was no convergence reflex of the pupil. Each optic disk was white, and the surface was retracted. A

small crescent of pale pink tissue was present at the upper nasal quadrant of the left disk, as had been observed before inoculation. The visual fields were as shown in chart 2. The patient was referred to the dermatologic clinic for further antisyphilitic treatment. The spinal fluid was not examined. The Wassermann reaction of the blood remained 4 plus.

Analysis of the records in cases 1 and 2 disclosed that the first patient said that he had never received any antisyphilitic medication, while the second patient had received an insufficient amount of treatment. In this regard some authorities consider malaria to be more effectual in combating syphilis of the central nervous system when little or no previous treatment has been given. Visual impairment was the first symptom observed by one patient, the onset of which dated one year before he

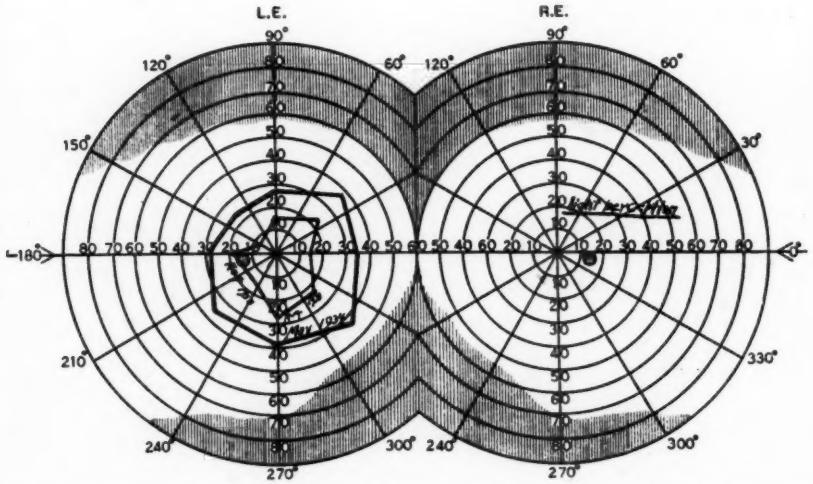

Chart 2 (case 2).—Visual fields as found in August 1933 and in May 1934. A 2 mm. test object was used at a radius of 330 mm.

presented himself in the ophthalmologic clinic. In the meantime he had accepted the advice of an optical salesman about the cause of his visual difficulties and had lost valuable time. There was no marked change in the visual acuity of either patient, but there were changes in the visual fields. The Argyll Robertson pupillary phenomenon, the reflexes and the serologic reactions were unaltered after treatment. The atrophic process of the optic nerves was far advanced before treatment was instituted. It was arrested by the treatment. A pink crescent was found on the nasal side of the left disk in one eye, and this eye obtained definite benefit from the treatment. I have made this observation in other patients and consider the finding of such a crescent in partial syphilitic atrophy to be a favorable indication.

CASE 3 (Tabes).—E. M., a white man aged 30, was admitted to the ophthalmologic clinic of the City Dispensary on Aug. 21, 1931, with the complaint of failing vision. He said that he had never had a venereal infection or treatment for a venereal disease. He observed that his vision for distance and for near objects was blurred early in the summer of 1931. The vision continued to decline. He could not read newsprint. Central vision in the right eye was 10/200 and in the left eye, 20/100. The left pupil was slightly larger than the right and about one half dilated. Both were immobile to light, and each one contracted during convergence. They expanded under 1 drop of 1 per cent homatropine. The fundi showed very pale and retracted disks. A small crescent of pale pink tissue was found on the nasal edge of each disk. The knee jerks were lost, and the Romberg sign was present. There was no impairment of speech. The visual fields were as shown in chart 3. The Wassermann reaction of the blood was 4 plus. Examination of the spinal fluid disclosed a 4 plus Wassermann reaction, no cells and a colloidal gold curve of 0122200000.

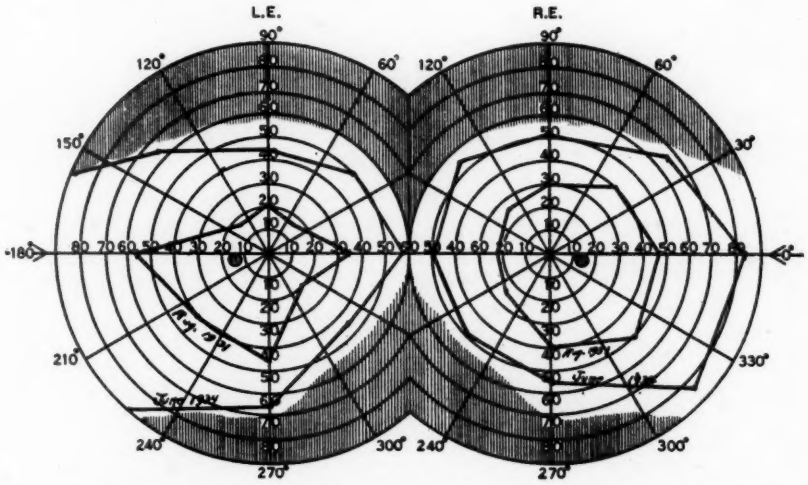

Chart 3 (case 3).—Visual fields as found in August 1931 and in June 1934. A 3 mm. test object was used at a radius of 330 mm.

The patient was sent to the hospital for inoculation with the organism of tertian malaria. There were fifteen paroxysms of chill with a temperature above 101 F. The highest temperature recorded was 105 F. There was increased color of the pink crescent on the nasal edge of the disk during the time malaria was in progress.

No antisyphilitic treatment of any kind was administered after malaria was terminated. The patient was examined at various intervals until June 1934, at which time vision in the right eye was 20/100 and in the left eye, 20/50. The pupils were round, equal, small and immobile to light stimuli but reacted during convergence. The fundi presented essentially the same appearances as were found at the time malaria was terminated. The visual fields were as shown in chart 4. The Wassermann reaction of the blood remained 4 plus. Permission to perform spinal puncture was refused. The knee jerks were abolished, and the Romberg sign was present. Speech was not impaired. The patient has not been seen since that date.

CASE 4 (Early Dementia Paralytica).—E. R., a white man aged 45, was admitted to the City Dispensary on Aug. 21, 1931, with the complaint of failing vision. The patient admitted that he had a chancre at the age of 24, but said that he had never received any treatment for syphilis. He noticed that his vision was impaired for newspaperprint early in 1931, and he had purchased glasses at different times from an advertising optometrist in a department store. The vision continued to decline. There was a positive Romberg sign; the knee jerks were exaggerated, and the speech was slurred. The pupils were very small, the left one being somewhat larger than the right; both were immobile to light stimuli, but contracted some during convergence. Central vision was 10/200 in the right eye; the left eye was blind. The fundi showed advanced primary atrophy of each disk. The Wassermann, Kahn and Kline reactions of the blood were each 4 plus. Examination of the spinal fluid disclosed a 4 plus Wassermann reaction; a cell count of 0; a trace of globulin, and a colloidal gold curve of 5554321000. The visual fields were found as shown in chart 5.

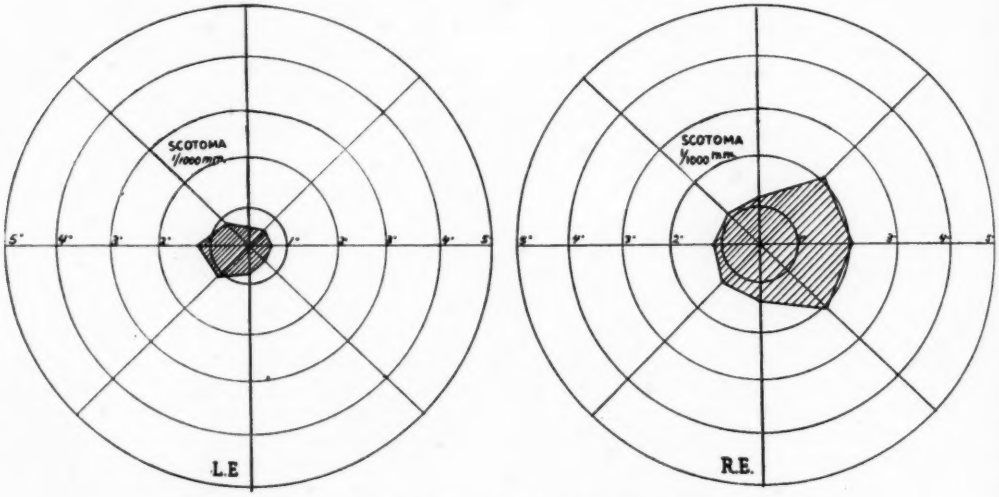

Chart 4 (case 3).—Central visual fields as found in June 1934.

The patient was referred to the hospital for inoculation with the organism of tertian malaria. Fourteen paroxysms of chill and fever with a temperature above 101 F. resulted. The highest temperature recorded was 104.6 F. There was subjective visual improvement during and after the malaria. No additional antisyphilitic drugs of any kind were given after malaria was terminated.

The patient was examined at intervals until July 1934, at which time the vision was 20/200 in the right eye; the left eye was blind. The pupillary reactions were unchanged. The field for the right eye was as shown in chart 6. Each fundus showed advanced primary atrophy of the disk. The spinal fluid disclosed a 4 plus Wassermann reaction, no cells or globulin and a colloidal gold curve of 3443221000. The knee jerks were exaggerated and the Romberg sign was present; the speech had lost the whine which formerly was present. The patient has not been seen since that time.

CASE 5 (Tabes).—H. S. B., a Negress aged 21, reported to the prenatal clinic of the Indianapolis City Hospital on Feb. 2, 1932. Gestation was of three months' duration. The Wassermann and Kline reactions of the blood were each 4 plus.

The patient was referred to the ophthalmologic clinic on account of the complaint of pain in each eye. Vision was 20/20 in both eyes. The pupils presented the

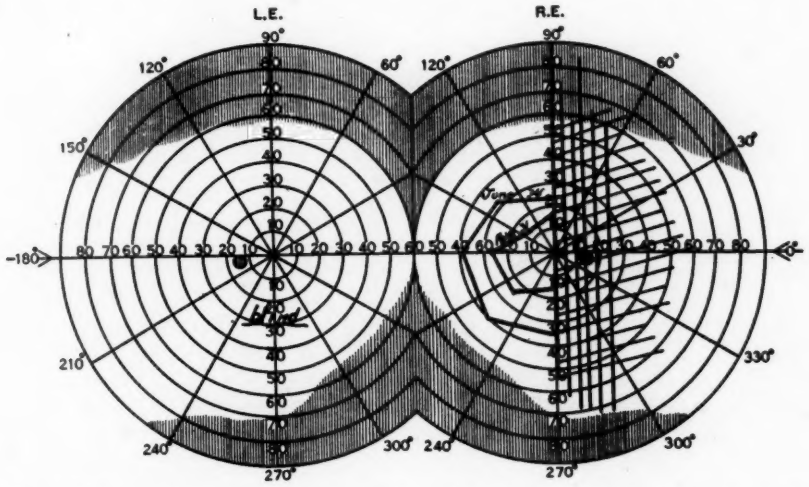

Chart 5 (case 4).—Visual fields as found in August 1931 and in June 1934. A 5 mm. test object was used at a radius of 330 mm.

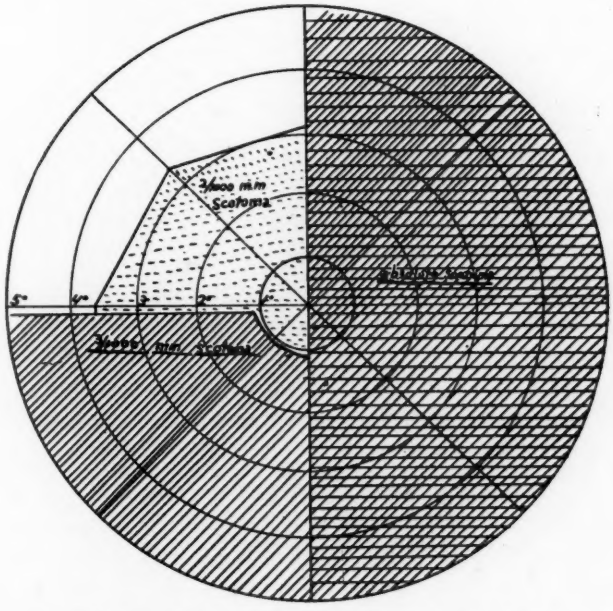

Chart 6 (case 4).—Central visual field as found for the right eye in June 1934.

Argyll Robertson phenomenon. Ocular motility was normal. The fundi were of normal appearance. The patient was referred to the dermatologic clinic for antisyphilitic treatment, where she received therapy with a bismuth preparation or with neoarsphenamine each week.

The patient appeared again at the ophthalmologic clinic on June 6. In the meantime she had miscarried at about the sixth month. She was unable to see and could not recognize large objects about the room. Examination disclosed perception of light present with the right eye. The left eye was blind. The pupils were widely expanded, equal in size, round and immobile to light stimuli. Ocular motility was unimpaired. The ocular fundi showed marked paleness and retraction of each nerve head. There was a small crescent of pale pink tissue on the upper nasal edge of the right optic disk. The arteries were contracted and threadlike in appearance. There were no other changes in the fundi.

The patient was referred to the hospital for artificial induction of malaria. It was found that she was resistant to the strain of the organism of tertian malaria used for the inoculation, and efforts to induce this disease were unsuccessful. The patient had never had malaria previously. There was no strain of the organism of quartian malaria available to which the Negro is susceptible; so it was necessary to resort to some other form of treatment.

Typhoid vaccine was administered intravenously on alternate days. The first dose consisted of 25,000,000 killed organisms suspended in several cubic centimeters of physiologic solution of sodium chloride. The succeeding doses were increased approximately 50 per cent.

A chill with elevation of body temperature from 102.5 to 105 F. occurred after each injection of the vaccine. Six injections were given. At the close of the treatment the patient could identify large objects with the right eye, such as the furniture in the hospital ward. There were no changes observed in the reaction of the pupils or the appearance of the ocular fundi except a possible increase in the intensity of the color of the upper nasal crescent of the right optic disk.

In August 1932 a field for the right eye was plotted on the tangent screen. Vision was 20/200 in the right eye; the left eye was blind. The pupils were widely expanded and immobile to light stimuli. The patient was returned to the dermatologic clinic for continuance of the antisyphilitic treatment, which had been discontinued at the time of her miscarriage.

The patient was reexamined at various intervals. On May 5 the central vision was 20/100 in the right eye; the left eye was blind. The pupils were widely expanded, round, equal and immobile to light stimuli. The left eye was slightly divergent. There was a narrow crescent of pink tissue present on the upper nasal edge of the right disk. The two optic disks were otherwise marble white, and the surface of each was moderately retracted.

On June 15, central vision in the right eye was 20/70; the left eye was blind. The right pupil was one half dilated and immobile to light stimuli. The left pupil was widely expanded and immobile to light stimuli. The eyes were unchanged otherwise.

On October 5 central vision was 20/70 in the right eye; the left eye was blind. The pupils and fundi were unchanged. The field of the right eye was found as shown in chart 7. Antisyphilitic treatment had been received since August 1932, which consisted chiefly of injections of neoarsphenamine and bismuth and mercury preparations. The Wassermann and Kline reactions of the blood were 4 plus. Examination of the spinal fluid disclosed a 4 plus Wassermann reaction, 5 cells per cubic millimeter, a colloidal gold curve of 01111100000 and a faint trace of globulin. The knee jerks were diminished; there was a questionable Romberg sign.

Cases 3, 4 and 5 present interesting features. One patient was resistant to the strain of the organism of tertian malaria used at the

institution. It is not unusual to find the Negro resistant to strains of the organism of tertian malaria but susceptible to strains of the organism of quartian malaria. Intravenous injections of typhoid vaccine were substituted. There was improvement in the vision of one eye of this patient which was phenomenal when one considers the apparent hopeless condition to which the vision had been reduced. The patient is able to go about unaided, whereas before she was dependent on other persons. The two white men were benefited by the induction of malaria. They received no other antisyphilitic treatment. The vision of both was improved three years afterward, and they were likewise

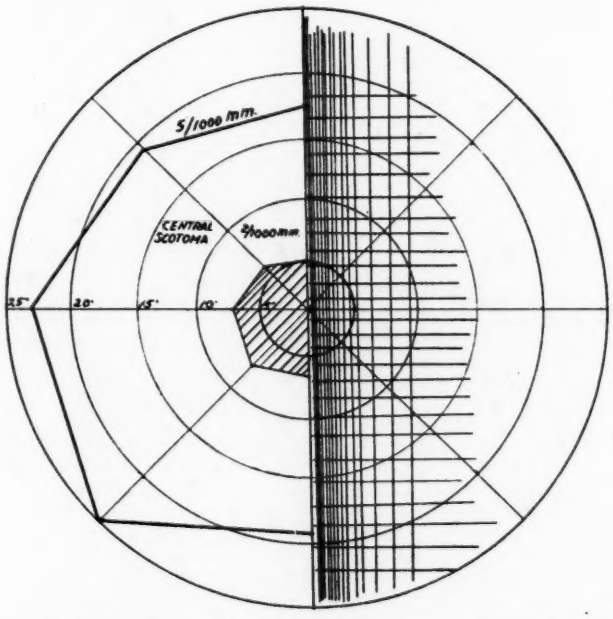

Chart 7 (case 5).—Visual field as found for the right eye in October 1934.

improved physically. The older one of the two was benefited the least. Age may have been a factor in producing changes in the circulatory system which affect adversely the response of the patient to malaria. Furthermore, the older one may have had syphilis for a much longer time. The state of the pupils was unchanged. The pink crescent on the nasal side of the disk was observed again. One patient was studied during a paroxysm of chill and fever. There was increased vascularity of the optic disk. No comparison is made between the production of malaria and intravenous injections of vaccine, and no conclusions are drawn concerning the relative value of the two in treatment of syphilis of the central nervous system. Typhoid vaccine was used as a substitute for malaria for the reason already given.

Case 6 (Dementia Paralytica).—W. C., a white youth aged 18, had congenital syphilis. The patient's father contracted syphilis before the child was conceived. The father died of dementia paralytica. The patient had received antisyphilitic treatment for several years. There was some notching of the teeth, roughness of the anterior edge of the tibia, a high palate and a flattened bridge of the nose. He had used glasses since early childhood and had been unable to read newspaperprint without them. In 1928 he was employed in the dyeing department of a hosiery mill. He began to have trouble with the vision of the left eye for small objects and had difficulty in comparing colors. The vision of the left eye gradually became worse. Later, the vision of the right eye became seriously impaired. In 1930 he suffered from a "nervous breakdown." He was admitted to the psychopathic ward of the Indianapolis City Hospital with the diagnosis of dementia paralytica. The Wassermann reaction of the blood was 4 plus. Examination of the spinal fluid showed: globulin, 3 plus; 14 cells per cubic millimeter;

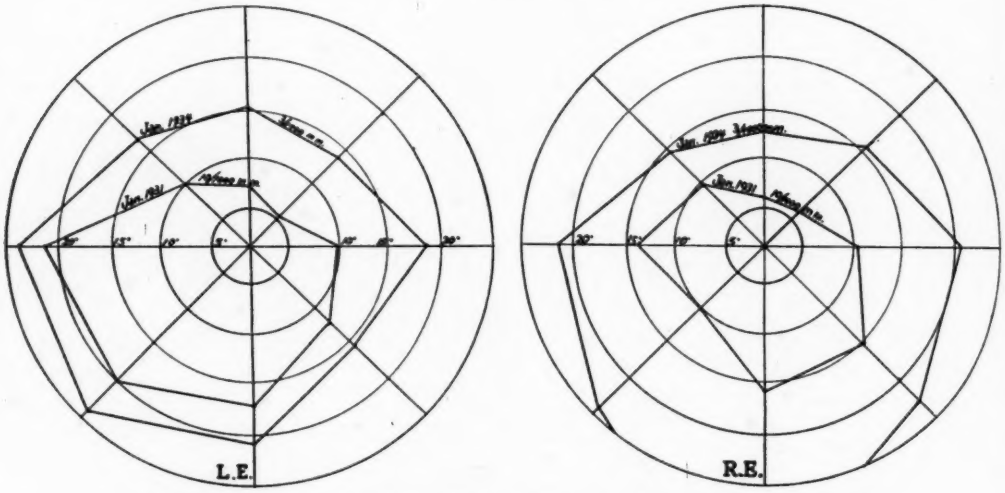

Chart 8 (case 6).—Visual fields as found in January 1931 and 1934.

colloidal gold curve, 5554444411, and Wassermann reaction, anticomplementary. The knee jerks were present; there was a suggestive Romberg sign, and the pupils were immobile to light but reacted in convergence. It was not my good fortune to examine the patient at this time, and the foregoing statements are taken from the hospital record. The central vision and visual fields had not been recorded.

The patient was inoculated with the organism of tertian malaria. There were thirteen paroxysms of chill and fever with the temperature above 101 F. The temperature crests varied from 103 to 105.2 F. and at one time was raised to 106 F. The patient became rational and made an excellent mental and physical recovery. A few months later I examined him in the ophthalmologic clinic. Central vision was 20/200 in the right eye and 20/100 in the left eye. The pupils were moderately dilated, equal and immobile to light and reacted well in convergence. The fundi presented a rather well advanced primary atrophy of the disks. The visual fields were as shown in chart 8. The patient returned to the dermatologic clinic, where he received injections of bismuth and mercury preparations and of neoarsphenamine.

I saw the patient at intervals during 1932, 1933 and 1934. There was some improvement in the central visual acuity. In May 1933 it was 20/100 in the right eye and 20/60 in the left eye. The January 1934 refraction in the right eye with a —0.25 sph. + 1.25 cyl., axis 85°, gave vision of 20/70; refraction in the left eye with —0.50 sph. + 0.75 cyl., axis 55°, gave vision of 20/30. The visual field was as shown in chart 8. The right pupil was round, and the left, obliquely oval; they were equal in size. Both were immobile to direct and consensual light stimuli and reacted in convergence. The ocular motility was normal. Examination of the fundus of the right eye showed the entire surface of the disk to be chalky white and retracted; the lamina cribrosa was visible over the central third; the vessels of the fundus were attenuated and straight. Along the larger arteries were many white streaks. The background of the macula and fundus showed no pathologic changes. The fundus of the left eye presented similar findings, except that there was a small crescent of pale pink tissue on the nasal side of the disk.

When the patient was seen in February 1935 the ocular condition was unchanged.

CASE 7 (Tabes).—E. C. T., a white man aged 46, first consulted me in 1926 for a change of glasses. He was an attorney and was a constant reader. He began wearing glasses at about the age of 14 and had used glasses ever since. With —3.50 sph. + 6.00 cyl., axis 115°, vision in the right eye was 20/30; with —3.50 sph. + 6.00 cyl., axis 85°, vision in the left eye was 20/30. A +2 sphere added for near vision enabled him to read Jaeger's test type no. 1. The pupils responded well to light and to convergence stimuli. The fundi were healthy. The blindspots were not enlarged on the tangent screen with the correcting lenses. The patient was advised to reduce his consumption of tobacco, which was considered excessive. He overindulged in alcoholic beverages and was advised to reduce or discontinue this habit.

The patient was reexamined annually until 1929. There was an interval from 1929 until 1932 when he was not seen by me. He was reexamined during April 1932. During the winter of 1931-1932 he noticed that his vision was worse, that he had great difficulty in distinguishing newspaperprint and that red and green traffic signals could not be determined with accuracy. He said that he had not used alcohol during the past two years and claimed to have used very little or no tobacco in the interval. During the preceding two months there had been some urinary incontinence. He had been unsteady when on his feet in a dark room. There had been some neuritis in the lower extremities. For the neuritis he had received a course of steam baths, and he felt relieved.

The knee jerks were abolished, and the Romberg sign was present. There was slight ataxia. The pupils were obliquely oval, equal in size and immobile to light stimuli and reacted well in convergence. Central vision with glasses was 3/60 in the right eye and 6/20 in the left eye. The optic disks presented the classic appearance of incomplete primary atrophy, the right disk to a greater degree than the left. The visual fields were as shown in chart 9.

The patient admitted having had a chancre ten years before. For this he received some intravenous injections of mercurial oil into the hips. He said that he had never had a cutaneous eruption. The Wassermann reactions of the blood and spinal fluid were each 4 plus.

The patient was inoculated with the organism of tertian malaria in August 1932 and had eight paroxysms of fever with a temperature above 101 F. The malaria was terminated after the eighth paroxysm on account of threatened cardiac failure. During the time that the malaria was in progress each optic disk was definitely hyperemic. There was no subjective change in the vision.

The patient returned to his home physician, who gave him courses of arsphenamine, a bismuth preparation and acetarsone.

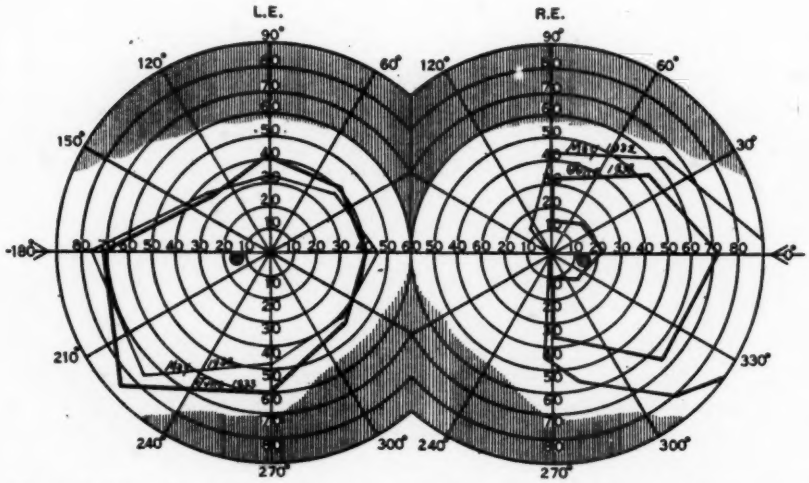

Chart 9 (case 7).—Visual fields as found in May 1932 and in June 1933. A 5 mm. test object was used at a radius of 330 mm.

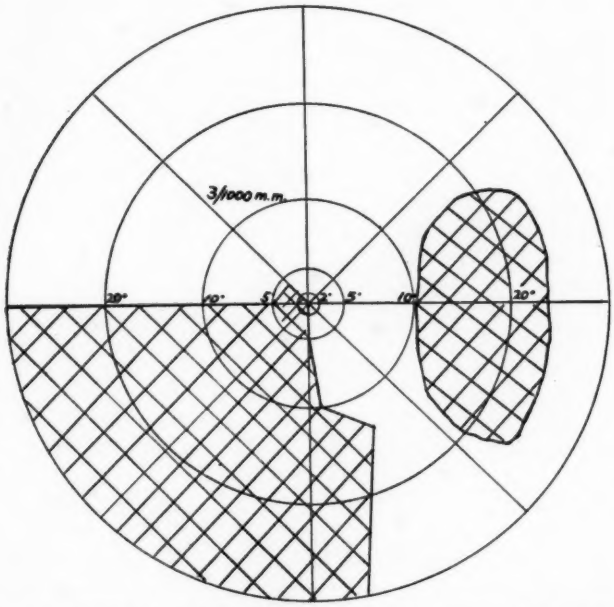

Chart 10 (case 7).—Central visual fields as found in May 1932.

In June 1933 central vision with glasses was limited to the temporal quadrant only in the right eye and was 6/30 in the left eye. The pupils were equal in size, round and immobile to light stimuli and responded well in convergence. The right optic disk showed a greater degree of atrophy than had existed one year

previously; the left disk was unchanged. The visual fields were as shown by chart 11. The patient said that he had had no neuritis and no disturbances of the bladder, had gained in weight and felt better.

Cases 6, 7 and 8 present certain features that should be emphasized. One patient had congenital syphilis. He had received a considerable amount of treatment. One cannot criticize the program of treatment. There was a period of approximately two years during which the vision of each eye was being impaired by syphilitic invasion of the optic nerves. After the mental symptoms developed and the patient had received treatment with malaria, the visual decline ceased and the invasion of the

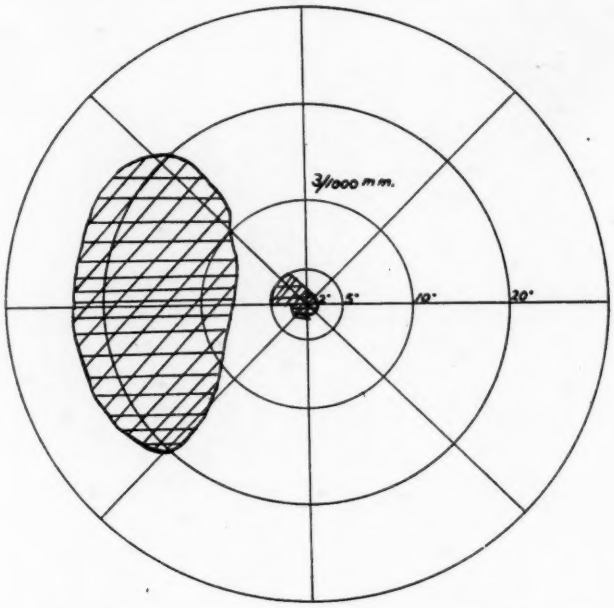

Chart 11 (case 7).—Central visual fields as found in June 1933.

nerve was halted. The case of the other male patient introduced the possibility of some damage to the optic nerve from the excessive use of tobacco and alcohol. This could not be proved or disproved, but certainly he did have syphilitic invasion of each optic nerve. He was benefited by malaria. The atrophic process was arrested. He was afterward placed on intensive treatment with metallic preparations. The first symptom shown by the female patient was impaired vision. She too had responded to the newspaper advertisement of a department store where poor vision, eyestrain and headaches were claimed to be relieved by glasses. She did not obtain benefit and lost valuable time. As a result, her vision is reduced to a degree greater than it might have been had treatment been given earlier. Malaria arrested the atrophic process

in the optic nerves. The serologic reactions and pupillary phenomena of these three patients were unchanged.

Case 8 (Tabes).—Mrs. H. E. G., a white woman aged 34, was first examined by me in November 1933. The vision had been steadily decreasing for the previous six months. She could not read newspaperprint and could not recognize persons across the street. She had obtained glasses from an advertising optometrist in a department store without benefit. She was told by him to have her teeth extracted. After all of her teeth were removed the vision continued to grow worse. She is the mother of two children, aged 16 and 18 years. She obtained a divorce in 1927 because her husband was immoral. She did not know when she became infected with syphilis. She had received no treatment, had had no cutaneous manifestations and did not know that she had syphilis until the ocular condition was properly diagnosed.

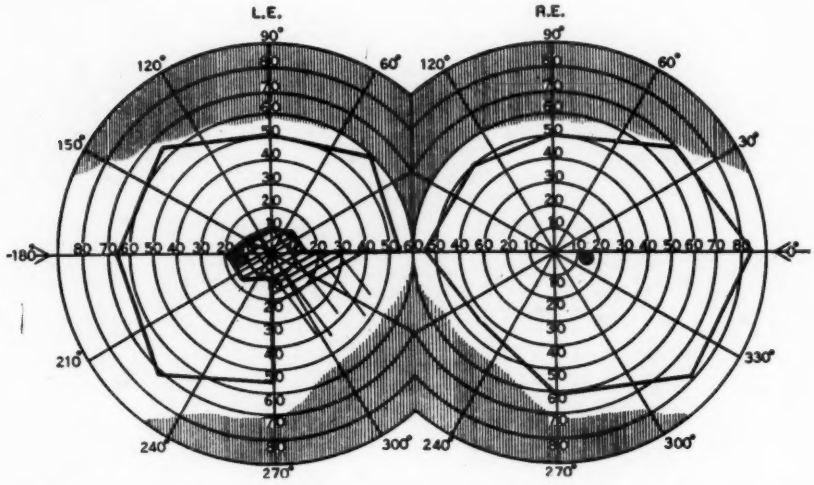

Chart 12 (case 8).—Visual fields as found in May 1934. A 5 mm. test object was used at a radius of 330 mm.

Central vision was 5/60 in the right eye and 1/60 in the left eye. The pupils were round and equal and responded very slowly to strong direct light and well in convergence. The fields showed a marked contraction to hand movements. The fundi showed advanced bilateral primary atrophy of the disks. The Wassermann reaction of the blood was 4 plus. The knee jerks were almost abolished. The Romberg sign was absent. There was no ataxia. The patient was inoculated with the organism of tertian malaria. There were eleven paroxysms of chill and fever. Two paroxysms gave a body temperature of 103 F., and nine, a temperature above 105 F. The optic disks showed a faint capillary vascularity.

In May 1934 central vision was 4/60 in the right eye and 1/60 in the left eye. The pupils were round and equal and reacted well to direct light and in convergence. There was a greater degree of atrophy of the left disk than was found at the first examination; the right disk appeared to be unchanged. The visual fields were plotted and are shown in chart 12. The patient said that she felt much better in general. She had received antisyphilitic treatment from her local physician after

malaria was terminated, which included neoarsphenamine, bismuth preparations and iodine bismuth compounds. A letter from her mother early in 1935 stated that her vision was unchanged.

CASE 9 (Tabes).—A. M., a white man aged 50, was admitted to the ophthalmologic clinic of the City Dispensary on Jan. 18, 1933. The Wassermann, Kahn and Kline reactions of the blood were 4 plus. The Wassermann reaction of the spinal fluid was 4 plus; the colloidal gold curve was 0123331000. The knee jerks were lost, and the Romberg sign was present. The patient observed impaired vision in September 1932. He had purchased glasses from a department store. He said that he had never had a venereal infection and had never received treatment for syphilis. Vision in the right eye was limited to perception of light; it was 20/100 in the left eye. The pupils were immobile to light stimuli but

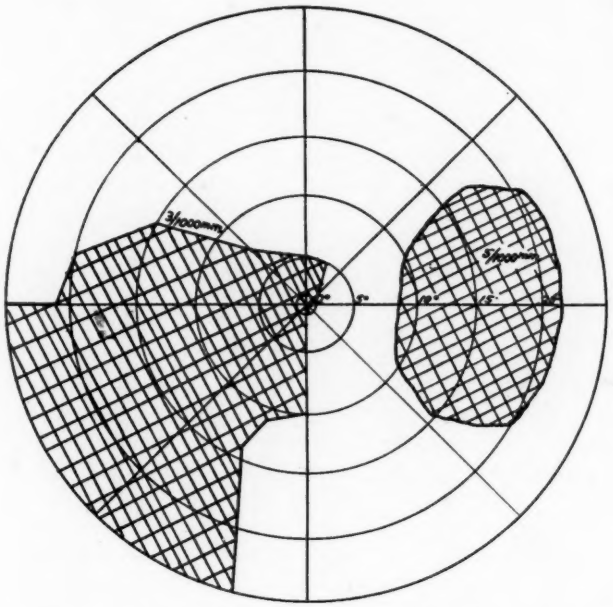

Chart 13 (case 9).—Visual field as found for the right eye in May 1934.

reacted well in convergence; the left pupil was larger than the right; both were obliquely oval. The fundi presented primary atrophy of the disks of an advanced degree. The patient was admitted to the hospital and inoculated with the organism of tertian malaria. Thirteen paroxysms of fever with a temperature above 101 F. resulted. In April 1934 the patient was blind. The disks showed advanced primary atrophy.

CASE 10 (Tabetic Form of Dementia Paralytica).—O. T., a Negress, aged 33, was admitted to the ophthalmologic clinic of the City Dispensary on May 1, 1932, with the complaint of blurred vision. She had received neoarsphenamine, and mercury and bismuth preparations for several months. The Wassermann and Kline reactions of the blood were 4 plus. The knee jerks were lost, and the Romberg sign was present. Vision was 5/200 in the right eye and 20/100 in the left eye. The pupils were unequal in size, the left one being larger than the right; they were immobile to light stimuli but responded well in convergence.

The patient was admitted to the hospital and inoculated with a strain of the organism of tertian malaria. There were two paroxysms above 101 F. The malaria stopped spontaneously. Additional intravenous treatment with vaccine was not administered. Quinine was given, and the patient was dismissed to the dermatologic department for further antisyphilitic treatment. Vision continued to be reduced. In August 1934 the right eye was blind, and shadows were perceived with the left eye. The Wassermann reaction of the spinal fluid was 4 plus; the colloidal gold curve was 4444410000, and the cell count, 4; globulin was present. The fundi presented advanced atrophy of the disks.

Case 11 (Tabes).—J. M. S., a white man aged 34, married, had a chancre in 1918, while in the United States Army. He received treatment from the medical department. After several months the Wassermann reaction of the blood was reported as negative on different occasions. He had received no antisyphilitic treatment after dismissal from the army.

The patient's wife had had no miscarriages. There were two apparently healthy children. In October 1930 the patient received a severe blow on the right side of the head. There was no fracture of the skull and no symptoms of concussion. He claimed that previous to this injury the vision of each eye was good and that he had no trouble of any kind. Four days later he noticed that the vision of the right eye was blurred, particularly for newspaperprint. There seemed to be a fog over all objects in the distance. The Wassermann reaction of the blood was 4 plus. The patient was given neoarsphenamine and a bismuth preparation. The children were examined. Each one had 4 plus Wassermann reaction and they were given antisyphilitic treatment.

Vision continued to fail, and the patient was referred to the ophthalmologic clinic on Feb. 6, 1931. Examination of central vision disclosed no perception of light in the right eye; vision was 20/70 in the left eye. The pupils were widely expanded, round and immobile to light stimuli. The fundi presented advanced primary atrophy of the disks, greater for the right than for the left. Inoculation with the organism of malaria was advised, but this was not done until May 22, 1931. In the meantime the patient had received intensive treatment with preparations of bismuth and mercury and with silver arsephenamine and neoarsphenamine. There were eleven paroxysms of fever with a temperature above 101 F. The highest temperature was 105.1 F.

On Aug. 2, 1934, the patient was blind. The pupils were widely expanded and immobile to light stimuli. The Wassermann reaction of the blood was 4 plus. The fundi showed complete atrophy of each disk.

Case 12 (Tabetic Form of Dementia Paralytica).—T. T., a white man aged 58, on May 19, 1933, reported to the ophthalmologic clinic on account of impaired vision. He had experienced difficulty in reading four months before and had obtained glasses from a department store without benefit. He said that he had never had venereal disease. The knee jerks were lost; the Romberg sign was present, and there was no disturbance of speech. The Wassermann and Kline reactions of the blood were 4 plus. There was no central vision in the right eye and vision was limited to perception of light in the left eye. The pupils were moderately dilated, round, equal in size and immobile to light stimuli. The convergence reflex was present. The fundi showed advanced primary atrophy of each disk. The patient was admitted to the hospital, and malaria was artificially produced on June 6. Twelve paroxysms of fever with a temperature above 101 F. occurred. Quinine was administered on July 25.

In October 1934, the patient was blind. The Wassermann reaction of the blood was 4 plus; examination of the spinal fluid disclosed a Wassermann reaction of 4 plus and a colloidal gold curve of 5542444440. The ocular fundi were unchanged. The pupils were moderately dilated, round, equal and immobile to light stimuli.

Cases 9, 10, 11 and 12 are cited as examples of unavoidable atrophy of the optic nerve when the treatment is given late in the course of the disease. The patient's vision was almost abolished before malaria was induced. The syphilitic process had completely invaded the nerve, had infiltrated through an entire cross-section of it or had impaired the circulation of the nerve to such a state that death of the part was inevitable. One Negro was incompletely treated with malaria and had a natural resistance to the parasite. Further treatment with vaccine administered intravenously was not carried out by the medical department for reasons not understood by me. Another patient contracted syphilis twelve years previously. He had received antisyphilitic treatment until the blood gave a negative reaction. After that he was not under medical supervision until the vision became impaired. This was noticed after an injury to the skull. This injects the question of what effect trauma may play in initiating syphilitic optic atrophy. It is my opinion that in this instance trauma played no part and had nothing to do with the onset or progress of the invasion and atrophy of the nerve. The last patient was reduced to blindness before he was examined and treated. Here again valuable time had been lost by the patient seeking advice from incompetent nonmedical sources.

SUMMARY

The records of twelve patients are presented. Each patient had syphilis of the central nervous system with syphilitic invasion of the optic nerve and visual pathways. There were eight patients with tabes and two each with dementia paralytica and dementia paralytica of the tabetic form. The condition of eight patients improved under therapy and in four there was no improvement. One of the eight patients was treated by injection of typhoid vaccine intravenously because she was resistant to the strain of the organism of tertian malaria used for that purpose at the hospital; the other seven were treated with induced malaria. There was no improvement in four of the patients, and the ocular condition continued to progress until blindness resulted. This was to be expected owing to the advanced state of atrophy of the optic nerve that was present before treatment with malaria was started.

The state of the pupils was unchanged in all twelve patients. All persons infected with syphilis should be carefully observed for any indication of invasion of the central nervous system. Attention should be paid to any invasion of the optic nerves and visual pathways, and if

these structures show evidence of involvement it would be prudent and wise to treat the patient as early as possible with malaria. The physician should remember that malaria does not possess magic power to restore function to an optic nerve with advanced atrophy of the tissue, but in order to employ the treatment efficiently, malaria should be induced early and thus enable the natural barriers to syphilis to be established before vital tissue has lost its function.

The action of therapeutic malaria in various forms of syphilis of the central nervous system has been considered to be due to different reactions of the body against malaria which are also inhibitive or destructive to the spirochete. I am of the opinion, as are others, that the elevation of body temperature has received too much emphasis in the literature on this subject. The elevation of body temperature must play a part in the defense exhibited to the spirochete, but it is questionable if much importance can be given to this factor alone in the treatment of syphilis of the central nervous system. Elevation of body temperature has not given results according to the height of the temperature or the number of paroxysms of fever. It has been pointed out that patients with dementia paralytica have shown improvement after the afebrile form of malaria. It is true that laboratory animals can be rendered free from spirochetal infection when the body temperature is elevated to a height greater than the human being can withstand, but this fails to support the claim that elevation of body temperature alone is the principal factor in the defense.

Stimulation of the reticulo-endothelial system by Plasmodium is the method of producing increased resistance to the spirochete of syphilis. Here, too, we are combating the invasion of one disease by another of a similar biologic level. Also, the shower of the organisms of malaria in the blood stream stimulates the reticulo-endothelial system the same as would the sudden intravenous liberation of any colloidal substance. The system of histiocytes, clasmatocytes and macrophages are stimulated. These cells are the important ones in the defense against the inroads of syphilis of the central nervous system. The response of the macrophages is greatest in the leptomeninges. These various defense cells have the peculiar property of arresting syphilitic infiltration of tissue. It is this peculiar tissue infiltration or invasion that characterizes the pathologic process of dementia paralytica. It is the same syphilitic infiltration to which Ingvar referred in his masterful work on the Argyll Robertson pupillary phenomenon. The optic nerves, chiasma and optic tracts are involved by the same meningeal infiltration which is largely responsible for the damage to those structures. When this infiltration is arrested before maximum damage has occurred, the decline of vision is halted and the patient is saved from blindness.

Additional factors probably play a minor rôle in the defense developed against the spirochete after malaria: vasodilatation and increased metabolism. It has been shown for some patients that there is a mild hyperemia of the optic disks during the paroxysm of malaria.

The factors of fever, vasodilatation, increased body metabolism and stimulation of the reticulo-endothelial system, with emphasis on the last factor, appear to be the means by which malaria and other acute febrile diseases offer to the body an effective defense against syphilis of the central nervous system.

EPITHELIAL DOWNGROWTH INTO THE ANTERIOR CHAMBER FOLLOWING CATARACT EXTRACTION

ARREST BY RADIUM TREATMENT

DERRICK VAIL, M.D.
CINCINNATI

The purpose of this paper is to consider the nature of downgrowth of epithelium and its clinical recognition and to point to a rational and simple plan of treatment which has proved to be successful in a few cases. Idiopathic and traumatic cysts of the iris will not be considered as pertinent to the subject, except as they may be influenced by the treatment to be discussed.

Collins and Cross, in 1892,[1] reported two cases in which epithelium was found lining the anterior chamber after cataract extraction. Glaucoma occurred eight months after operation in the first case, and when the eye was removed, two years later, it showed laminated epithelium on the back of the cornea, the anterior surface of the iris, the capsule of the lens and the hyaloid membrane. Apparently this is the first case on record. The authors presented two theories regarding the mode of formation of these cysts: (1) The epithelium arose from proliferation of the endothelium lining Descemet's membrane and the anterior surface of the iris and (2) surface epithelium was carried in at the time of operation and subsequently proliferated. They inclined toward the latter theory, which is now considered the correct explanation, and argued in support; "besides the improbability of any other source for the epithelium of the cysts than that of implantation from the outer surface of the eye, there is the positive evidence that epithelial lined cysts have been produced in this way experimentally in rabbit's eyes." They undoubtedly had in mind the experiments of van Dooremaal[2] (1873), Goldzieher (1873)[3] and Hosch[4] (1885), wherein bits

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Read at the Seventy-First Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 7, 1935.

1. Collins, E. T., and Cross, F. R.: Two Cases of Epithelial Implantation Cyst in the Anterior Chamber After Extraction of Cataract, Tr. Ophth. Soc. U. Kingdom **12**:125, 1892.

2. van Dooremaal, J. C.: The Development of Living Tissue Implanted into Foreign Soil, Arch. f. Ophth. **19**:3 and 359, 1873.

3. Goldzieher, cited by Villard and Dejean: Iris Cysts, Arch. d'opht. **50**:91, 1933.

4. Hosch, F.: Experimental Studies on Iris Cysts, Virchows Arch. f. path. Anat. **99**:446, 1885.

of conjunctival epithelium implanted into the anterior chamber of animals led to epithelial cysts.

Collins,[5] discussing the same subject in 1914, remarked that had he examined his earliest patients by means of serial sections he undoubtedly would have found epithelial extension from the wound.

Guaita,[6] in 1893, described the histologic picture in an eyeball which had healed slowly following cataract, in which iridocyclitis occurred ten months later. On clinical examination there had been observed a semitransparent membrane of peculiar appearance on the anterior surface of the iris and over the pupillary area. On histologic examination this membrane was seen to be stratified epithelium (three layers) on the posterior part of the corneal scar extending over the iris, forming with Descemet's membrane a sort of biconvex sac. Guaita was probably the first to point out the relationship between downgrowth of epithelium and a slowly healing wound.

Meller [7] (1901), according to Parsons,[8] was the first to give a comprehensive report. Many authors, notably Elschnig,[9] Morax and Duverger [10] and Speciale-Cirincione,[11] have since written on this subject. particularly in regard to the gloomy prognosis and the histologic picture in cyst of the anterior chamber.

Since cyst of the anterior chamber, if allowed to progress, eventually destroys the eye through severe iridocyclitis and glaucoma, a gloomy prognosis is justified. Any measure, therefore, which offers the slightest chance to arrest or retard the development of cyst of the anterior chamber must be seriously considered.

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5. Collins, E. T.: Discussion on Post-Operative Complications of Cataract Extractions, Tr. Ophth. Soc. U. Kingdom **34**:21, 1914.

6. Guaita: Proliferation of the Corneal Endothelium on the Iris and the Pupil Space After Cataract Extraction, Arch. d'opht. **13**:507, 1893.

7. Meller: Epithelium in Anterior Chamber After Cataract Extraction, Arch. f. Ophth. **52**:3 and 436, 1901.

8. Parsons, J.: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1904, vol. 1, p. 165.

9. Elschnig: Epitheliation of the Anterior and Posterior Chamber as a Cause of Glaucoma After Cataract Extraction, Klin. Monatsbl. f. Augenh. **41**:1 and 247, 1903.

10. Morax and Duverger: Epithelial Invasion of Anterior Chamber, Ann. d'ocul. **141**:1, 1909.

11. Speciale-Cirincione, F.: Glaucoma Provoked by Epithelial Investment of the Anterior Chamber Following Cataract Operation, Atti. d. Cong. Soc. ital. oftal., 1924, p. 180.

Cysts of the iris have been successfully removed surgically. Berg,[12] Eversbusch,[13] Elschnig and Grossmann[14] have reported spontaneous absorption of cyst of the iris, which Michail[15] believes can be explained by spontaneous autolysis of the epithelial cells. Wright[16] cured a patient with cyst of the iris by the injection of phenol into the cyst, followed by repeated washing out of the sac with saline solution. Thilliez[17] (1908) and Chaillous[18] (1914) employed electrolysis with complete success in cases of traumatic cyst of the iris. None of these methods of treatment would seem to apply to the cases in which epithelium lined the anterior chamber, although it is conceivable that electrolysis might be effective in this condition.

The clinical diagnosis of downgrowth of epithelium offers great difficulties. E. Fuchs[19] stated that "the formation of an epithelial lining for the anterior chamber cannot be diagnosticated clinically, since the epithelium is transparent." This teaching has been and is still widely held by ophthalmologists. Yet Collins (1914) remarked that he had made such a diagnosis clinically which he verified by pathologic examination. Others who have made the diagnosis prior to enucleation or pathologic verification are: Custodis,[20] Fazakas,[21] Michail,[15] Salus[22] and Meller[23] (1923).

Considering the enormous number of operations for cataract performed by operators of different ability and practicing various methods, the rather rare cases of epithelial downgrowth indicate that special conditions must exist for the normal epitheliation of the corneal wound

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12. Berg: Cyst of Anterior Chamber, Operation, Klin. Monatsbl. f. Augenh. **65**:879, 1920.

13. Eversbusch: Spontaneous Recession of an Iris Cyst, Klin. Monatsbl. f. Augenh. **31**:409, 1893.

14. Grossmann, cited by Villard and Dejean: Iris Cysts, Arch. d'opht. **50**:91, 1933.

15. Michail, D.: Epitheliation of Anterior Chamber of Eye Following Extraction, as Revealed by Biomicroscopic Examination, Ann. di ottal. e clin. ocul. **60**: 553, 1932.

16. Wright: Cyst of the Iris, Brit. J. Ophth. **9**:454, 1925.

17. Thilliez, cited by Villard and Dejean: Iris Cysts, Arch. d'opht. **50**:91, 1933.

18. Chaillous, cited by Villard and Dejean: Iris Cysts, Arch. d'opht. **50**:91, 1933.

19. Fuchs, E.: Textbook of Ophthalmology, ed. 8, Philadelphia, J. B. Lippincott Company, 1924, p. 620.

20. Custodis, E.: Epithelial Lining of Anterior Chamber After Cataract Extraction, Klin. Monatsbl. f. Augenh. **89**:612, 1932.

21. Fazakas, S.: Epitheliation of Anterior Chamber of Eye After Operation, Orvosi hetil. **76**:776, 1932.

22. Salus, R.: Healed Epitheliation of the Anterior Chamber, Klin. Monatsbl. f. Augenh. **78**:368, 1927.

23. Meller, T. J.: Ophthalmic Surgery, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1923, p. 225.

to go awry. Corrado [24] has attempted to explain by experiments on animals how this occurs. He found that it was not sufficient for a strip of epithelial tissue to happen to fall into the anterior chamber in order to have attachment and proliferation occur. He showed that only when the corneal wound remains open beyond a certain time is it possible for the corneal epithelium, by proliferation, to penetrate into the anterior chamber after having covered the border of the wound itself. One of the causes which facilitate this development is the incarceration of the iris, the capsule of the lens or the vitreous in the corneal wound. When the capsule of the lens is entangled the epithelium finds a surface well adapted to its growth and spread. The smoother the surface of implantation, the more easily the epithelium tends to proliferate along that surface. Corrado also found in his experiments that in those eyes in which there was resorption of the strip of epithelium introduced into the anterior chamber the resorption was more delayed in the eyes in which he had succeeded in maintaining a general condition of hypotony. He believes it possible, therefore, that an aqueous humor rich in proteins, as is the case when the anterior chamber has not completely reformed, is a medium better adapted for the nutrition and thus the vitality of the tissue introduced.

Corrado's experiments support the well known clinical fact that epitheliation of the anterior chamber occurs, as in the majority of the cases reported, when there has been a slow healing of the corneal wound, hypotony or detached choroid, with delayed formation of the anterior chamber.

The development of epithelial downgrowth may be extremely rapid, as in Meller's [7] (1901) case, in which both sides of an extraction wound were covered with epithelium in four days, or slow, as in Morax and Duverger's [10] and Papolczy's [25] cases, occurring from four to ten years after operation. Papolczy believes that epithelial growth progresses so long as the wound is open and stops when it is closed. This belief is aided by the observation of Custodis [26] (1933), who found that the epithelium of a traumatic cyst of the iris proliferated much more rapidly after opening of the anterior chamber and attempted removal of the cyst.

A proper study of the histology of the disease process is necessary as a diagnostic and therapeutic aid. Parsons described this as follows:

> On reaching the back of the cornea the epithelium insinuates itself between Descemet's membrane and substantia propria and also grows on the posterior

24. Corrado: Glaucoma Secondary to Post-Operative Penetration and Proliferation of Epithelium into the Anterior Chamber of the Eye, Ann. di ottal. e clin. ocul. **59**:706, 1931.

25. Papolczy, F.: Partial Epitheliation of the Anterior Chamber After Cataract Extraction, Klin. Monatsbl. f. Augenh. **84**:266, 1930.

26. Custodis, E.: Contribution on the Pathology and Treatment of Iris and Iris Chamber Cysts, Klin. Monatsbl. f. Augenh. **90**:361, 1933.

surface. A variable number of layers of cells are found, the lowest cells being cylindrical or cubical, the highest very flat and long. The same applies when the iris is covered or the whole chamber lined; the epithelium varies extremely in thickness, from a single layer to five or six or more and the cells are found ill formed. Sometimes they can be distinguished from endothelial cells only with difficulty.

Meller noted many goblet cells which discharged their contents into the anterior chamber. Urmetzer [27] also mentioned goblet cells and was the first to give the name "anterior chamber cyst." Collins noticed that many cells were swollen and mucoid. Custodis mentioned that there were many degenerated ones producing "little pearls." Speciale-Cirincione found new epithelial cells formed of abundant protoplasm with a small nucleus. Guaita, staining some of his sections with the Gram stain, observed the presence of new formations of karyokinetic shape. Papolczy noted that the individual epithelial cells were smaller as a rule than the corneal epithelium and the nuclei stained strongly. Michail mentioned the dropsical cells, which were calciform, with voluminous protoplasm. The nuclei were basophilic. He attributed the slowness of development in his case to autolysis of the cells.

It should also be borne in mind that epitheliation of the posterior surface of the iris and lens occurs (Elschnig,[9] Nagano [28]). This may have a bearing on the success or failure of radiation treatment.

In order to make a clinical diagnosis of lining of the anterior chamber by epithelial cells, one must first have this condition in mind. A slowly healing wound, delayed formation of the anterior chamber, a leaking aqueous and fistulization of the wound are potential precursors. If, in addition, one discovers a thin haze on the posterior surface of the cornea, beginning at the wound and extending downward as time goes on, the possibility of this being epitheliation is no longer remote. Repeated careful and studied examination with the slit lamp and microscope reveals certain features which can make the diagnosis reasonably certain. Four biomicroscopic descriptions have been found in the literature. These are as follows:

1. In the case reported by Custodis the upper half of the posterior surface of the cornea and the entire iris was covered as with a veil, spotted with minute pearls. The edge of the veil on Descemet's membrane was moundlike. The gray-green iris looked as though it had been covered with milk, suggesting the picture of the conjunctiva in vernal catarrh. It should be easier to see on a brown iris than a gray one.

27. Urmetzer, cited by Ginsberg, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 1, p. 548.

28. Nagano: Epitheliation of Anterior and Posterior Chamber After Cataract Extraction, Arch. f. Augenh. **65**:116, 1909.

This author commented that one can see the membrane on an after-cataract only when the latter is very thin and the pupil is black, as Salus was able to do. The degenerative cell changes, noticed by most observers pathologically, should be recognized with the slit lamp and the corneal microscope. In this case these were the "little pearls" and looked like bubbles of air in water glass on the wall of the membrane.

2. In the case reported by Fazakas there was a fine cloudy layer of tissue on the posterior surface of the cornea, with the convexity downward, and on the anterior surface of the iris and upper part of the coloboma, with the convexity upward. The layer removed from the upper part of the coloboma proved to be one of epithelial cells.

3. In the case reported by Michail the anterior surface of the iris was covered on its free part with a fine transparent membrane blending with the membrane of the secondary cataract, passing to the posterior surface of the cornea and extending beyond the synechia of the iris. (This was proved histologically.)

4. In the case reported by Salus a thin, pigment-studded membrane was on the posterior surface of the cornea, forming a bow-shaped fringe bordering the entire width of the cornea. It covered the iris on both sides of the coloboma and pupillary space. The under-surface of the edge of the pupil showed a thin gray veil. This author was able to notice a gray membrane over a black pupil.

Frequent and repeated biomicroscopic studies in all these cases, as well as in my case, showed a slow and gradual development of the thin membrane on the posterior part of the cornea and more rapid development on the anterior surface of the iris, owing to its better nourishment here.

Reticular formations in the anterior chamber (Vogt [29]) and inflammatory membranes must be ruled out in the clinical diagnosis. These formations may be in all parts of the anterior chamber but are generally in the lower part or over the entire iris, as can be observed in chronic iridocyclitis. In downgrowth of epithelium, however, the membrane extends downward from the wound. "Reticular formations or pseudomembranes are at first connected with the surface of the iris, but they may ultimately become detached; on the other hand, in deep infiltrations of the cornea, the formation starts from the posterior surface of the cornea, from which it may become detached, forming a pedicle" (Koby [30]).

29. Vogt, A.: Atlas of Slitlamp-Microscopy of the Living Eye, ed. 2, Berlin, Julius Springer, 1930, pt. 1, p. 283.

30. Koby: Slit-Lamp Microscopy of the Living Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1930, p. 178.

Data for Nine Cases of Cyst of the Iris, Cyst of the Anterior Chamber or Downgrowth of Epithelium

Author	Case	Diagnosis	Duration	Type of Treatment	Number of Treatments	Dose*	Filter	Result	Comment
von Szily: Atlas der Kriegsaugenheilkunde samml. begleitendem Text, Stuttgart, Ferdinand Enke, 1916 cited by Custodis,[25] p. 391	1	.....	.....	.....	.....	.....	.....	.....	.....
Axenfeld: Ber. ü. d. Versamml. d. ophth. Gesellsch. **41**: 312, 1918	2	.....	.....	.....	.....	.....	.....	.....	No details
Jendralski: Klin Monatsbl. f. Augenh. **68**: 175, 1922	3	Cyst of anterior chamber	.....	Roentgen	1	30 min.; 30 cm. focus	4 mm. aluminum	Secondary glaucoma	Excision of cyst; phthisis bulbi; enucleation 4 mos. later; foreign body found surrounded by epithelial cells; roentgen treatment apparently without effect
	4	6 mo. old child; congenital cyst	From birth to 6 mos.	Radium	2, 5 wks. apart	26 mg. of mesothorium; 30-40 min.	0.4 mm. silver	Retrogression	Three mos. after treatment posterior surface of cornea slightly opaque; treatments stopped because of strong reaction of lids
Handmann: Klin. Monatsbl. f. Augenh. **72**: 111, 1924	5	Traumatic cyst of iris	2 years	Roentgen	4	20 min. each; 2 ma. load	3 mm. aluminum	Disappearance after 10 mos.	Cyst removed by iridectomy; recurred in 3 wks.; roentgen treatment started; vision improved from perception of movements of hands to 4/35

Handmann: Klin. Monatsbl. f. Augenh. 74: 218, 1925	6	Traumatic cyst of iris	4 yrs. after injury	Roentgen	6	10% E.D. on 5/1, 5/10, 6/1, 9/1, 9/14, 10/15; 29 cm. focus	3 mm. aluminum; hard rubber	Almost entire disappearance 6 mos.	Total dose about ½ epilation; from 5/1 to 9/1 slow growth; from 9/1 to 12/1 quick shrinkage
Salus,[21] 1927	7	Downgrowth of epithelium after cataract extraction	1½ years	Roentgen	2	70 E.D. each time	Not mentioned	Quick disappearance	Membrane grew rapidly after needling; sensitivity of cornea slowly returned; good vision; tension normal; patient died in 1931
Custodis,[19] 1932	8	Downgrowth of epithelium after cataract extraction	6 mos. after operation	Roentgen	1	100% E.D.; 30 cm. focus	0.5 mm. copper; 1 mm. aluminum	Iridocyclitis; enucleation 1½ yrs. later	Diagnosis confirmed by pathologic examination; roentgen rays had no effect; fistula in wound; chronic inflammation of iris and vitreous
Custodis,[25] 1933	9	Traumatic cyst of iris	1 year	Radium	1	20 mg.; 3 hrs.; 1 cm. focus	1 mm. lead	Further growth of cyst	Cyst punctured after radium treatment; surgically removed after first roentgen treatment;
				Roentgen	1	100% E.D.; 30 cm. focus	0.5 mm. copper; 1 mm. aluminum, 1/29/31	Cyst attained former size, 4/14/31	2 wks. later fine membrane quickly disappeared; lens remained clear up to 2 years later;
				Roentgen	2	50% E.D.; 30% E.D.; 30 cm. focus	0.5 mm. copper; 1 mm. aluminum, 1/29/31	Disappearance of film	No recurrences of membrane; vision 6/5

* E.D. indicates erythema dose.

A proliferating capsular remnant, especially when entangled in the wound, might conceivably simulate epithelium, but I believe that repeated careful biomicroscopic examination would disclose its true nature.

Striate keratitis and duplication or folds of Descemet's membrane should offer no particular difficulty in diagnosis with the slit lamp and corneal microscope. Vitreous herniation and prolapse into the anterior chamber present a definite picture which cannot be confused.

TREATMENT

Operation does no good but only produces a new flaring up of the process and increases epithelial growth. It is fruitless for combating the ensuing glaucoma (Custodis). An exception, however, is found in the report of a remarkable case by Fazakas: A fine layer was removed from the iris and proved by microscopic examination to be epithelium. After the separation of the membrane or "apron" of new tissue, this investigator cauterized (with the actual cautery) the margin of the freshly made wound and the inner surface of the upper part of the cornea. There was no recurrence of epithelial growth. Fazakas attributed the success of his treatment to the irradiation of thermal energy.

Irradiation with either the roentgen rays or radium offers hope in the cure or at least the arrest of epitheliation of the anterior chamber. Nine cases of cyst of the iris, cyst of the anterior chamber or down-growth have been reported in the literature. The details are to be found in the accompanying table, and it will be seen that the treatment was successful in seven of the nine cases. Fazakas' case, while not strictly included in the group, might be considered as one in which cure was produced by irradiation.

To determine the sensitiveness of different parts of the eye to the roentgen rays, Rados and Schinz [31] experimented on rabbits by giving small doses at short intervals over a considerable time. They concluded that, contrary to many reports in the literature, the eye is one of the organs of the body least sensitive to the roentgen rays. It is much less sensitive than the skin. The danger of production of cataract is very slight, and the lids can show not only epilation but also ulceration without the least change being observed in the cornea, lens or retina. These investigators determined the depth penetration of 150 per cent. of a skin erythema dose [32] in the eye by means of the absorption curve in water, with a Wulf single thread electrometer and large ionization chamber.

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31. Rados and Schinz: Experiments on Animals with Regard to Ocular Sensitivity to Roentgen Therapy, Arch. f. Ophth. **110**:354, 1922; Treatment and Healing of Carcinoma with Roentgen Rays, ibid. **110**:370, 1922.

32. A skin erythema dose is the dose of roentgen rays which produces a slight erythema of the skin after eight days and a browning after three weeks.

Suzuki [33] studied the effects of the roentgen rays on implantation cysts experimentally produced in the anterior chamber and iris of the cat. He found that the roentgen rays (from 1 to 3 erythema doses) produced degeneration of the epithelial cells of the anterior wall of the cyst. The epithelial cells of the posterior wall remained unaffected. Prophylactic treatment of the implantation membrane was without influence on the development of the cyst.

REPORT OF A CASE

A woman, aged 66, had glaucoma absolutum in the left eye following a dislocated cataractous lens. The eye was removed because of pain in 1928, at which time an immature cortical cataract was found in the right eye. This had become mature by Sept. 12, 1933, at which time an intracapsular cataract extraction was performed, with the Knapp-Stanculeanu forceps method, fortified by the Verhoeff (no. 1) suture, the Van Lint lid akinesia and suture of the superior rectus muscle. The operation proceeded smoothly and without complications. The healing, however, was stormy, the wound remaining open, and the anterior chamber was not reformed for fourteen days. This was attributed to a persistent squeezing of the lids. The conjunctiva was congested and edematous, and striped keratitis persisted for more than three weeks. The patient was discharged on the sixteenth postoperative day. The pupil was low; the pillars of the coloboma of the iris were apparently free, and there was a good red reflex, but the wound was slightly bulged. Ciliary congestion lasted several weeks.

On November 4, vision with a + 9.0 sph. $\supset$ + 4.0 cyl. ax. 60 was 20/70. The eyeball was somewhat congested, and a haze in the posterior portion of the cornea was noticed above at the edge of the wound. This haze or membrane developed downward on the iris as well, so that on November 29 it had extended to within 2 mm. of the lower edge of the pupil. The intra-ocular tension was found to be 35 mm. (Schiötz). A 2 per cent solution of pilocarpine hydrochloride was ordered to be instilled four times daily. The tension returned to normal (20 mm., Schiötz).

On Jan. 17, 1934, the edge of the membrane having advanced still farther on the posterior surface of the cornea and the anterior surface of the iris, the patient was referred to Dr. Charles Goosmann, a radiologist of Cincinnati, with the diagnosis of epithelial downgrowth into the anterior chamber. He used a radium plaque 5/8 inch square (1.6 cm. square), containing 15 mg. of radium and covered with a very thin monel metal protecting screen, with further filtration through two thicknesses of rubber stock, a total of about 1 mm. of rubber. This was applied directly on the eyelid over the area involved and left on for two hours (fig. 1).

On January 25, the tension was 18 mm. (Schiötz), and the use of pilocarpine was discontinued. With the slit lamp it was noted that the upper third of the cornea was slightly edematous; a few vacuoles were seen in the epithelium and nasal side of the cornea. These were not present before and were attributed to the effects of radium, as in a similar case described by Vogt.[34] The edge of the new

33. Suzuki, E.: The Therapeutic Effect of Roentgen Ray on Implantation Cyst of the Iris (Experimental Study), Acta soc. ophth. jap. **38**:553, 1934; abstr. Zentralbl. f. d. ges. Ophth. **32**:507, 1934.

34. Vogt,[29] p. 258.

gray membrane on the posterior surface of the cornea was seen as a thicker irregular white line. The upper two thirds of the iris and pupillary space were covered with a diaphanous grayish membrane, stippled with fine dots of pigment in the pupillary area. The margin of the pupil beneath the membrane was slanted backward. The nasal pillar at the angle was drawn forward and was adherent to the posterior surface of the cornea by a reflected band of the membrane.

On March 20, no further progress in the membrane growth was noted. The tension was 18 mm. (Schiötz). The membrane appeared less distinct and the pupil blacker. The edge of the membrane on the posterior corneal surface and across the pupillary area was more sharply defined. The red reflex was brighter. Vision with a + 9.0 sph. ⊃ +3.0 cyl. ax. 60 was 20/40 + 2.

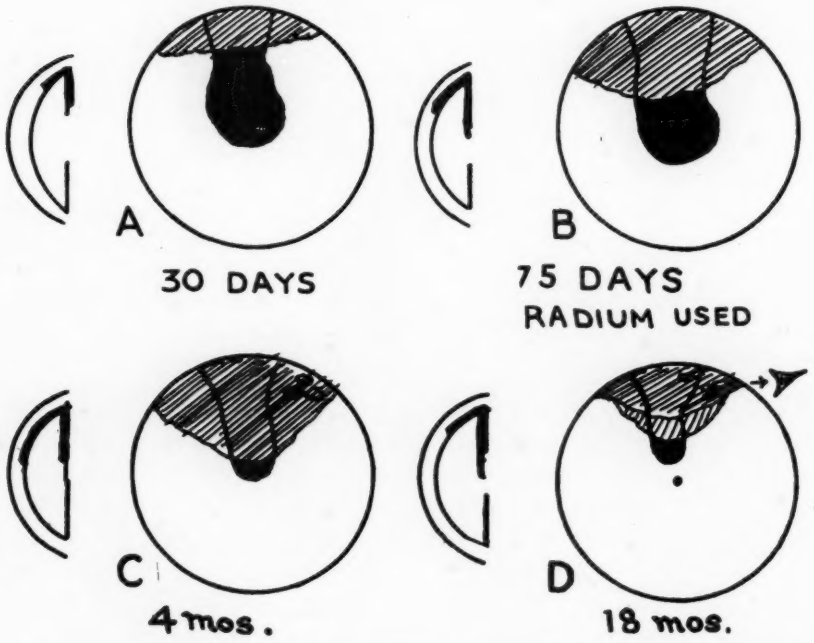

Fig. 1.—*A*, growth of membrane on the posterior surface of the cornea and the anterior surface of iris thirty days after cataract extraction; *B*, appearance seventy-five days after operation, when radium was used; *C*, appearance four months after operation; *D*, appearance eighteen months after operation.

On April 13, the slit lamp showed a few more vacuoles in the superficial epithelium of the cornea, which were seen best with transmitted light. The pupillary opening was somewhat rounder.

On November 24, vision was unchanged. The tension was normal. The slit lamp showed no further downgrowth on the cornea or iris. This could roughly be measured by the length of the slit beam of light. Vacuoles were still present. The pupillary opening was smaller, probably owing to the pulling upward of the iris. The epithelium of the cornea over the gray film was somewhat bedewed.

On March 5, 1935, vision (with the same correction as previously) was 20/30 —2; the pupillary opening was very small. The membrane was definitely thinner.

except for the edges, the distinctness of which remained about the same. The pupil was slightly less than 1 mm. in diameter. The tension was 18 mm. (Schiötz). The cornea was practically insensitive above but was normal below. The slit lamp showed a slight bedewing of the corneal epithelium above, but the larger vacuoles had disappeared. The membrane was obviously thinner, and that on the cornea was of much less extent, especially on the temporal side. There was anterior synechia of the iris at from 10 to 11 and from 1 to 2 o'clock. The thin sheet of tissue extended over this and reflected onto the posterior surface of the cornea with adherent strands.

On April 8, the slit lamp showed no vacuoles in the epithelium (fig. 2). The downgrowth looked like a bubble and as though a transparent hammock were slung between the anterior synechiae of the pillars of the iris. The lower edge of the membrane over the pupillary space was double refractive and smooth. The tension was 20 mm. (Schiötz). Vision (with the same correction as previously) was —20/30 (every letter). The upper half of the cornea, which had remained insensitive, now began to be sensitive, although it was still not normal. It could not be determined whether this was the result of the operation or of radium treatment.

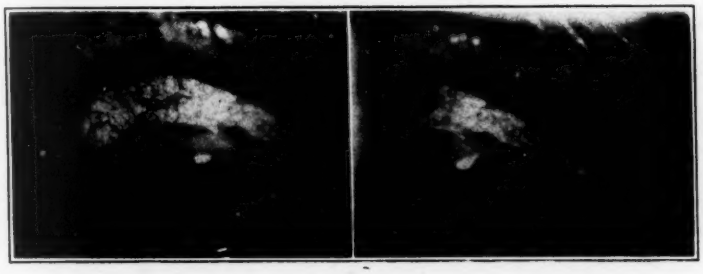

Fig. 2.—Epithelial downgrowth into the anterior chamber in Vail's case.

COMMENT

Two problems need to be solved in considering this subject. The first is the diagnosis of the epithelial downgrowth in the living eye. I believe that such a diagnosis can be made by careful and repeated biomicroscopic studies, especially in all cases of glaucoma following cataract extraction. The essential point seems to be a cloudy veil developing down from the wound onto the posterior portion of the cornea and anterior surface of the iris. In a patient with a brown iris and with intracapsular extraction (black pupil), as in the present case, the membrane could be easily seen. It might be difficult in cases in which the iris is gray or blue and much secondary cataract substance remains.

The second problem is that of treatment. In most of the cases reported in the past the condition has been recognized as epitheliation of the anterior chamber only after enucleation and pathologic studies. Apparently all the eyes had been enucleated because of absolute glaucoma and pain. Operation for glaucoma was of no avail, except in the case reported in this article. On the contrary, it appears to increase

the growth of cells. Irradiation, either with the roentgen rays or with radium, offers some chance of arresting, at least, the spread of the process.

Much work, particularly in the form of statistical studies, needs to be done to decide several points. The chief point to be settled is whether the roentgen rays or radium is the better agent. It appears, from a study of the few cases reported, that the roentgen rays are the more effective. The second point is the determination of the proper dosage and method of application. It must not be forgotten that epitheliation of the anterior chamber is a destructive lesion and calls for drastic treatment, regardless of its danger to the eye. Experiments have been carried out which indicate that the roentgen rays are not harmful to normal ocular structures if properly applied. The available evidence indicates that the newly developed epithelial cells are destroyed by the roentgen rays more easily than malignant cells and that the latter are more easily affected than normal epithelium.

819 Carew Tower.

DIAGNOSIS AND TREATMENT OF ANISOPHORIA

JONAS S. FRIEDENWALD, M.D.
BALTIMORE

Anisophoria is defined as that form of heterophoria in which the degree of muscular imbalance of the eyes varies with the direction of gaze. The existence of motor anomalies of this type is well known to the medical profession, but their analysis as a special group and their systematic treatment by optical means has not heretofore been attempted. For this reason it seems not inappropriate to segregate this particular group under a new name. In general, anisophoria is known to occur in two groups of cases: (*a*) The first group comprises cases in which the heterophoria is due to paresis or spasm of one or more of the extraocular muscles. In these there may be an average overall amount of heterophoria which may be corrected with prisms, leaving a smaller or larger amount of uncorrected heterophoria of opposite sign in opposite quadrants of the field of view. (*b*) The second group comprises cases in which anisophoria is produced by the correction of anisometropia with lenses which have a different peripheral prismatic effect for each eye.

For instance, a patient with paralysis of the right superior rectus muscle may exhibit, after almost complete recovery, a left hyperphoria of 2 diopters on looking downward, a left hyperphoria of 4 diopters on direct gaze and a left hyperphoria of 6 diopters on looking upward. If this patient is provided with prisms which correct the average overall hyperphoria of 4 diopters there will remain an amount of muscular imbalance which will vary with the direction of gaze; i. e., increasing right hyperphoria on looking downward and increasing left hyperphoria on looking upward.[1] Since, in this case the right eye tends to rotate through a smaller angle on vertical movement than the left eye, one may express this by the designation of right vertical microphoria. Finally, if the deviation in the optical axes of the two eyes is symmetrical in upward and downward rotation, and is proportional to the gross amount of

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From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

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1. The exact amount of residual heterophoria depends to some degree on the base curve of the correcting prism and on whether it is placed base up before the right eye or base down before the left eye, for the effective power of a prism varies with the direction of gaze.

rotation of the two eyes away from the axis of direct gaze, the degree of anisophoria may be specified as so many prism degrees of anisophoria per so many arc degrees of rotation.[2]

This method of designating the amount of the anisophoria may be illustrated by the hypothetic case of paresis of the right superior rectus muscle already referred to. Suppose that after correction of the overall left hyperphoria of 4 diopters the patient has orthophoria in direct gaze, 1 prism degree of right hyperphoria on looking down 10 degrees, 2 prism degrees of right hyperphoria on looking down 20 degrees, 1 prism degree of left hyperphoria on looking up 10 degrees, 2 prism degrees of left hyperphoria on looking up 20 degrees, etc. It is evident that this patient has a right vertical microphoria of 1 prism degree per 10 degrees of vertical rotation of the eyes. Since 1 prism degree is equal to one half an angular degree of rotation, this condition may be expressed as 5 per cent right vertical microphoria. It will be shown later that in most instances it is convenient to measure the degree of anisophoria at some specified angular deviation from the axis of direct gaze, and that an angular deviation of 20 degrees of rotation away from the axis is in general the most convenient one to use. One may use the results of such measurements directly to designate the amount of anisophoria. Thus, in the case already mentioned, the patient shows 4 prism degrees of right vertical microphoria in the total range of 40 degrees of vertical rotation from a downward gaze of 20 degrees to an upward gaze of 20 degrees. We may write this as: right vertical microphoria 4 prism degrees per 40 degrees of rotation, or more simply, since unless otherwise specified the measurements will always be made at 20 degrees off axis—that is, for a total rotation of 40 degrees—one may designate this merely as 4 degrees of right vertical microphoria. From what has been said it is evident that the expressions "4 degrees of right vertical microphoria" and "5 per cent right vertical microphoria" are equivalent.

METHOD OF MEASUREMENT

For the crude determination of the existence of anisophoria it is often sufficient to tip the patient's head backward or forward, to one side or to the other, in the routine measurement of heterophoria for distance or near. Such crude measurements, however, have the disadvantage that the angle of deviation of gaze is not readily measurable and also that, if the patient has even a moderate degree of horizontal muscular imbalance, and if in tipping his head backward and forward the movement is slightly oblique, the measurement of the vertical phoria will be considerably in error. For this reason it was decided to develop a simple apparatus in which several muscle lights would be arranged, the central one being directly in front of the patient, while the other lights would be placed

2. In this paper the symbol Δ will be used to designate prism degrees instead of prism diopters while the symbol ° will be used to designate arc degrees of rotation.

at definite distances away from this light and in line with definite meridians of rotation of the eyes.

Since, in anisophoria, the degree of muscular imbalance increases as the eyes are rotated farther and farther away from the position of direct gaze, it is obvious that very small amounts of anisophoria can best be determined by making the measurements with the eyes directed as far off axis as possible. A few preliminary tests indicated that 20 degrees off axis was the maximum deviation that could be conveniently used. In the first place, this represents the limit of the field of ocular rotation ordinarily used in those scanning movements of the two eyes not associated with movements of the head. In the second place, while most patients can perform ocular rotations of considerably greater amplitude than this, fixation often becomes unsteady when the limits of rotation are approached, and very considerable degrees of heterophoria of no clinical significance may suddenly become apparent in these extreme positions. Finally 20 degrees is, roughly, the angular difference in the position of the eyes for vision at distance and for reading. For these reasons an angular deviation of 20 degrees was chosen for the routine measurement of anisophoria. Occasionally smaller degrees of deviation may be useful.

A rod 3½ feet (106.6 cm.) long subtends an angle of 20 degrees for an observer 10 feet (3 meters) away. Since the sitting height of most people is about 4 feet (122 cm.) it is not simple to measure the phoria for downward rotation of the eyes by 20 degrees at a distance greater than 10 feet. To make the measurement at 20 feet (6 meters) would require that the patient be put on a pedestal or that the muscle light be placed 3 feet (91 cm.) below the floor. These considerations led to a choice of 10 feet as the maximal distance at which anisophoria was to be measured.

The apparatus for the measurements at this distance is shown in figure 1. It consists of a rod, 7 feet (2.1 meters) long with a small electric light at each end and at the center. The rod is placed in the plane perpendicular to the axis of direct gaze with the central light directly in front of the patient. The rod may be rotated in such a way that the lights at each end may be placed in any meridian of ocular rotation which one desires to test but always lie just 20 degrees away from the axis of direct view. The patient is directed to hold his head quite still and to look first at one light and then at another. The degree of heterophoria for any direction of gaze may then be measured with a phorometer. The Maddox rod cannot be used in such a test, because the line of light appears bent when one looks obliquely through the rod.[3] The Stevens phorometer can be used without any difficulty, but it is desirable to tip the prism into a position perpendicular to the direction of gaze in order to avoid a variation in their effective power.

There is every reason to believe that the degree of anisophoria in many cases would be different if the measurements were made at a considerable distance with convergence relaxed and again close by with convergence active. It is therefore desirable to test the anisophoria both for distance and for near. The instrument

3. If one desires to use a Maddox rod in this test, this objection can be eliminated by having the rod bent in its long axis, so that when it is placed in the phorometer the center of curvature of the long axis of the rod will coincide with the center of rotation of the patient's eye. The portion of the rod through which he is looking will then always be perpendicular to the optical axis of his eye, and the line of light will appear straight no matter what the position of the muscle lights.

already described which is used at 10 feet from the patient is quite adequate for distance testing, since the degree of accommodation and convergence required is minimal. In order to test anisophoria for near the same type of instrument in a diminutive model may be used. In fact, since with the phorometer the images of the two eyes are disassociated, one might use the same apparatus and merely put minus lenses before the patient's eyes, forcing the eyes to accommodate and therefore also to converge. While some interesting observations can be obtained in this way, it is difficult to be sure that accommodation is actually being maintained, and this does not seem satisfactory for routine tests.

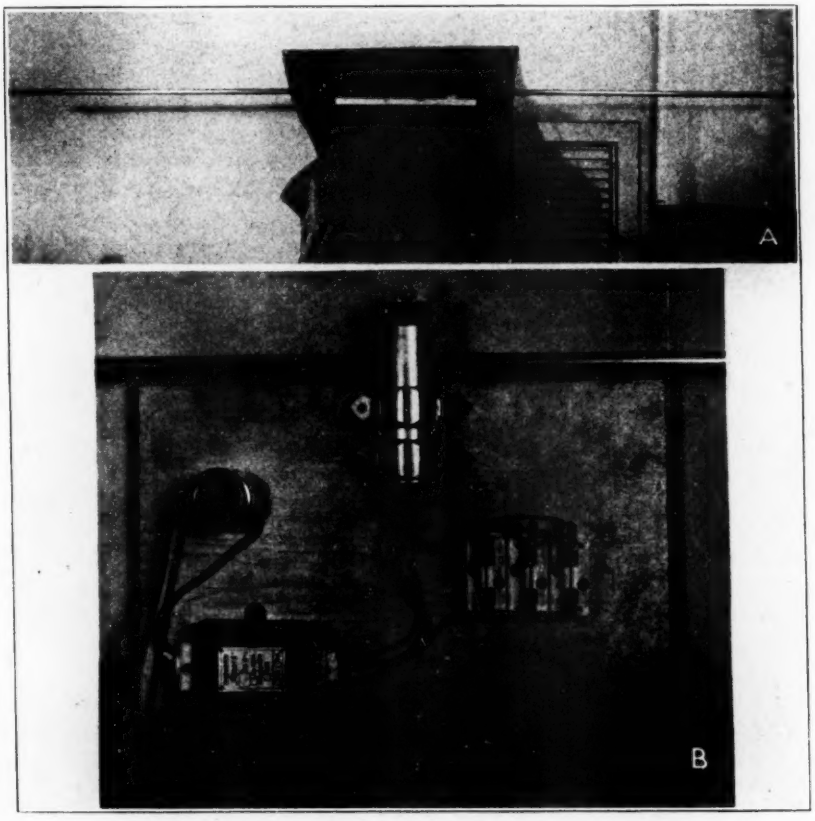


Fig. 1.—Anisophorometer for distance: *A*, front view; *B*, top view.

The apparatus which I have used for testing anisophoria for near has been based on the ingenious method of disassociating the images of the two eyes which Ames developed in the construction of his aniseikonometer. The instrument is shown in figure 2. It consists of a shallow box with its front made of a sheet of sand-blasted aluminum, the surface of which is sufficiently mat to avoid any stimulus to fusion. The box is placed directly in front of the patient at a distance of 12 inches (30.5 cm.) from the plane of his pupils. Directly in front of the patient in the center of the aluminum sheet there is a small pinhole, and similar holes are bored 4½ inches (11 cm.) above, below and on each side of this central hole. There are also similar holes at a distance of 4½ inches from the central hole in each of the oblique quadrants. With the instrument properly placed

before the patient, the distance of any of these holes from the center subtends an angle of very nearly 20 degrees for each eye of the patient. Behind each hole there is a small light which is set so that the right eye of the patient sees each hole as a bright spot, while the left eye sees only a black hole. These images are so disparate that they cannot be fused, and the degree of heterophoria in each direction of gaze can be measured quite simply with prisms. Sometimes in the use of this instrument the disassociation of the images can be facilitated by placing a red glass before the right eye.[4]

PHYSIOLOGIC ANISOPHORIA

Horizontal Anisophoria.—Several facts about the variations in horizontal heterophoria with variations in the direction of gaze are already well known. A few additional findings have been elicited in the course of the more detailed analysis of these phenomena. There is first

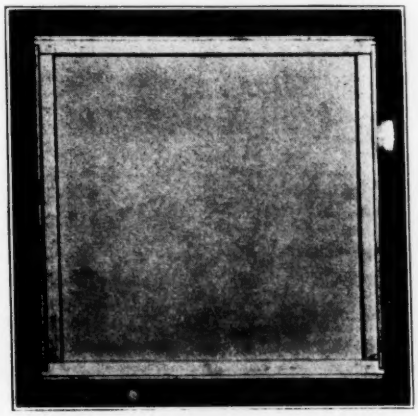


Fig. 2.—Anisophorometer for near.

the usual decrease of esophoria or increase of exophoria with convergence. With upward gaze, as is well known, there is increasing exophoria; with downward gaze, increasing esophoria. In many normal persons there is a slight tendency toward esophoria on lateral gaze, especially for near.

Vertical Anisophoria.—There is, in general, no normal variation in vertical heterophoria with the direction of gaze at distant objects. In otherwise normal persons slight variations in vertical heterophoria are, however, frequently found when the eyes are directed far out in the oblique directions. These variations seem to depend on slight imbalance between the actions of the superior or the inferior rectus muscle

4. Dr. Adelbert Ames (personal communication) has pointed out the necessity, in measuring anisophoria with prisms, of bearing in mind the variation in the power of the prism when the direction of gaze is toward its apex or base. The amount of this variation will be different in simple prisms and in rotating double prisms, etc.

of one eye and the inferior or the superior oblique muscle of the other. When these slight variations are present in otherwise normal and symptomless patients they are usually symmetrical, indicating a general overaction of the vertical recti in relation to the obliques, or vice versa. I have not been able to determine which of these two states is most common.

When one comes to consider the vertical muscular balance for near it becomes at once obvious that inequality in the vertical rotation of the two eyes is necessary if binocular single vision is to be maintained over any extended area in near vision. Figure 3 shows the visual axes of the two eyes when a vertical arrow is seen nearby and somewhat to one side of the sagittal plane. Since the arrow is closer to the patient's left eye than to his right eye it subtends a greater angle at the center of rotation of the left eye than at that of the right, and the left eye

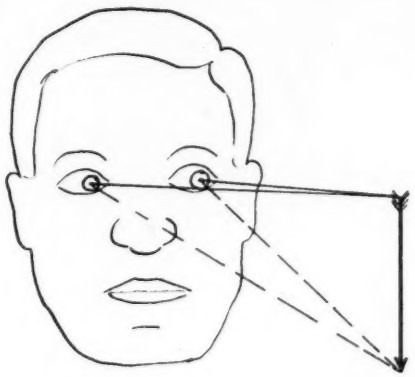


Fig. 3.—Physiologic anisophoria for near.

must rotate farther downward from the horizontal plane than the right eye when the gaze is directed toward the bottom of the arrow. Any patient who, when tested for phoria at the near range, shows no disparity in the visual lines in the oblique positions must, therefore, be making greater vertical movements of the abducted eye. The disparity in vertical movement required is by no means insignificant. For instance, when the patient looks at one of the obliquely located points in the instrument for testing anisophoria at near range described, the adducted eye makes a vertical rotation of 16½ degrees while the abducted eye makes a vertical rotation of 17½ degrees. The abducted eye must therefore make 6 per cent greater vertical movement in order to arrive at this direction of gaze than the adducted eye, and if this were not regularly accomplished in normal persons one could regularly measure up to 2 prism degrees of vertical heterophoria in the oblique positions for near. As a matter of fact, the variations found in these positions show

the same small irregularities found in the vertical heterophoria for distance in the oblique positions, the adducted eye making sometimes a little more, sometimes a little less, than the required movement.

It is interesting to compare the accuracy with which these disparate vertical movements are normally carried out with the tendency toward esophoria in near lateral gaze already mentioned. When the eyes are directed from a near point straight ahead to another point in the same frontal plane but somewhat to one side, the abducted eye must make a greater horizontal excursion than the adducted eye. If this is not accurately performed, i. e., if the two eyes tend to make the same lateral excursion, esophoric displacement will result, and this is precisely what has been found in many normal persons.

Tortional Anisophoria.—The present study has not included tests for cyclophoria, and I have, therefore, nothing to add to the well known variations in cyclophoria with near and far and upward and downward gaze.

PATHOLOGIC ANISOPHORIA

As mentioned, pathologic anisophoria may result either from unequal activity of the extra-ocular muscles of the two eyes or from unequal peripheral prismatic action of lenses prescribed for the correction of anisometropia. In the former case the anomaly may be due either to paresis or to spasm of one of the extra-ocular muscles. In such cases there is usually an overall heterophoria, and the anisophoria becomes manifest only when the overall heterophoria has been corrected by prisms. In some instances, the overall factor may be lacking. When this factor has been discovered it is generally found that the anisophoria is a fairly stable element of the ocular movements, though, like other forms of heterophoria, it is subject to some variations in amount. The symptoms of anisophoria are as multiple as those of other forms of asthenopia, but a rather large percentage of patients refer their discomfort to one eye only, complaining especially of pain, lacrimation and photophobia. In fact, from the short experience I have had with the study of this condition it appears that any patient who after accurate correction of ametropia and of the ordinary heterophorias complains persistently of discomfort in one eye only is a likely candidate for anisophoric correction. In those with large amounts of anisophoria vertigo and the other heterophoric symptoms are likely to occur.

Vertical or oblique anisophoria have in my experience been much more productive of symptoms than has horizontal anisophoria. The latter may cause some confusion in spacial judgments but, unless present in such high degree as to be associated with strabismus of paralytic type, it does not, as a rule, cause asthenopic symptoms. In the vertical or oblique meridian, however, quite small degrees of anisophoria may occasion discomfort. Thus, a patient who has only 0.5 prism degree of

vertical heterophoria in the extremes of the usual upward and downward gaze may have as severe symptoms as one who has an overall and unvarying heterophoria of double this amount. Naturally all patients do not react equally to their ocular difficulties and the severity of symptoms is hard to evaluate, but one gets the impression in studying cases of this type that the relative severity of the symptoms produced by an anisophoric disturbance as compared with those produced by an equal overall and unvarying heterophoria is much the same as the relative severity of symptoms produced by astigmatism compared with those produced by an equal amount of spherical ametropia.

The least amount of anisophoria which I have so far thought was causing symptoms has amounted to 1 prism degree of anisophoria in a total of 40 degrees of vertical movement of the eyes. In such a case there is just a little over 1 per cent difference in the vertical movements of the two eyes. The least amount of anisophoria measurable with the apparatus described is somewhat less than half this amount.

There are at least three optical methods for the correction of anisophoria. The first two are simple but of somewhat limited applicability; the third is somewhat more complex but of more general applicability.

1. Patients with anisophoria whose symptoms are wholly or largely due to the heterophoria from which they suffer in downward gaze, as in reading, may be relieved by inserting a prismatic reading segment into their glasses or by having them use separate reading glasses which contain prismatic correction for the average degree of heterophoria on downward gaze. Presbyopic patients who are already wearing bifocal glasses or separate reading glasses are most easily helped by this method. It is plain, however, that whereas the heterophoria varies with the direction of gaze, the effect of the prism is approximately uniform over its whole area and that this device merely reduces the amount of anisophoria in one portion of the field, leaving the heterophoria undercorrected or overcorrected to a varying degree depending on the direction of gaze. Nevertheless, symptomatic relief may be given to a substantial number of patients in this way, and this device has been widely used by many ophthalmologists in the past. It is, in fact, the only method that ophthalmologists have had in the past for dealing with this condition.

2. An even simpler measure, one which patients frequently discover by themselves but which I have not found mentioned in the ophthalmologic literature, is that of holding the reading matter somewhat toward the macrophoric side in cases of vertical or overall anisophoria. From what I have said it is clear that when a patient looks toward the lower right hand corner of a printed page held before him he must make a greater downward movement of the right eye than of the left eye. Under normal circumstances these compensating deviations of the visual axes are made quite automatically. If, however, the patient has a right

hyperphoria in downward gaze, the compensating deviation will be performed with increasing difficulty as he looks down and to the right, while the opposite compensating deviation will be performed with increasing ease as he looks down and to the left. Precisely similar conditions will hold for the upward oblique movements, and the patient will therefore be much more comfortable if he holds his book a little to the left in reading or, what amounts to the same thing, turns his head a little to the right.

Many patients whose anisophoria results from the correction of an anisometropia automatically discover this position of maximum ease in reading. The reading matter is held well up, so as to escape the more marked degrees of heterophoria in the extreme directions of gaze, and a little to one side to equalize the rotational movements of the two eyes. The optimal required displacement is not as a rule very great and may be roughly computed as follows: One diopter of difference in refraction of the two eyes will produce 1 prism degree of heterophoria at 20 degrees of rotation off axis, or 2.5 per cent anisophoria. In order that an object held at the usual reading distance of 30 cm. may be 2.5 per cent closer to one eye than to the other it must be displaced about 4 cm. to one side of the midline. A simple rule to remember, therefore, in cases of anisometropia is to advise the patient who has discomfort in reading to displace his reading matter toward the more hypermetropic side by 1½ inches (3.7 cm.) per diopter of difference in the refractive correction of the two eyes.

The same maneuver is effective also in some cases in which the anisophoria is due to muscular anomaly. In case, for instance, the anisophoria is the result of a slight weakness of the right superior or of the right inferior rectus, the patient will find comfort in holding his book a little to the left, for in this position the anisophoria is not only partially compensated for but actually reduced. When, however, the anisophoria is due to a weakness of one of the oblique muscles, this fails, for the heterophoria increases as the paretic eye is adducted. In this case the patient tilts his head laterally but does not introduce any inequality in the distance of his eyes from the reading matter.

3. From what has been outlined it is obvious that an accurate optical correction of anisophoria can be obtained if lenses can be produced which possess no dioptric power but in which the peripheral prismatic action varies with the direction of gaze off axis. Such lenses are readily constructed. Any meniscus lens of no dioptric power will be found to show a peripheral prismatic action which increases regularly with the excentricity of the gaze, with the thickness of the lens and with the depth of the meniscus. It is possible, moreover, in lenses of actual dioptric power to vary the peripheral prismatic effect without altering the dioptric power by varying the depth of the meniscus and

the thickness of the lens, though the computations and technical complications in the development of such lenses are by no means readily solved. One may thus, within the limits fixed by the usual size of spectacle lenses and the possible variations in thickness, change the peripheral prismatic power of the patient's spectacle lenses without changing their dioptric power. Finally concavoconvex cylindric lenses may be combined with the patient's ordinary refractive correction in such a way as to correct meridional anisophoria. Ames and his coworkers [5] in developing lenses for the correction of aniseikonia have

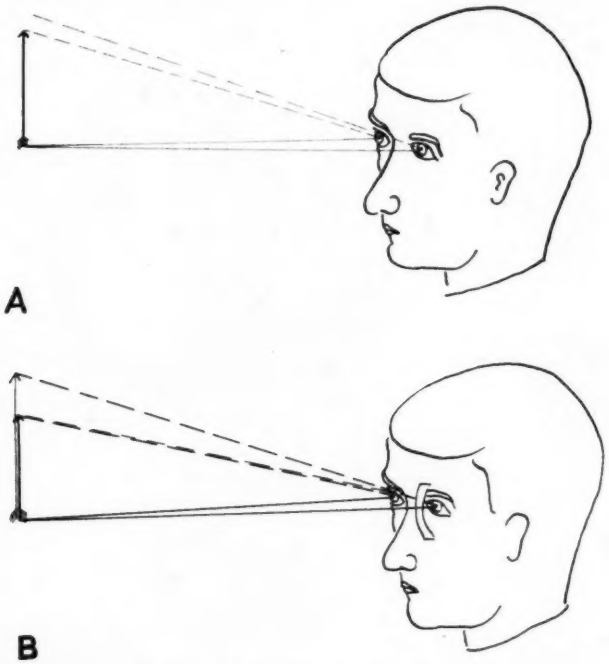


Fig. 4.—*A*, right vertical microphoria; *B*, right vertical microphoria corrected by enlarging the image for the left eye.

studied the mathematical and technological problems involved in the construction of lenses of these types in great detail and have at the same time computed the peripheral prismatic action of the lenses which they

5. Ames, A., Jr.; Gliddon, G. H., and Ogle, K. N.: Size and Shape of Ocular Images: I. Methods of Determination and Physiologic Significance, Arch. Ophth. **7**:576 (April) 1932. Ames, A., Jr., and Ogle, K. N.: III. Visual Sensitivity to Differences in Relative Size of Ocular Images of Two Eyes, ibid. **7**:904 (June) 1932. Ames, A., Jr.; Gliddon, G. H., and Ogle, K. N.: Ann. Distinguished Serv. Found. Optom. **1**:61, 1932; J. Optic Soc. America **22**:538, 1932. Ames, A., Jr., and others: Aniseikonic Lenses, Hanover, N. H., Dartmouth Medical School, 1934.

have designed. Their lenses can therefore be used quite readily for the correction of anisophoria.

The optical action of such meniscus lenses of zero power is shown diagrammatically in figure 4. Now it is clear that the peripheral prismatic action of lenses of this type is intimately associated with their magnifying power. In fact, if simple lenses of this type were free from spherical aberration the peripheral prismatic action would be precisely related to the magnifying power. However, all such lenses show in varying degree the phenomenon of barrel-shaped distortion; that is, the peripheral prismatic action and the peripheral magnification are somewhat less than the paraxial magnification. Furthermore, this statement is correct only for zero power lenses used at distance. With

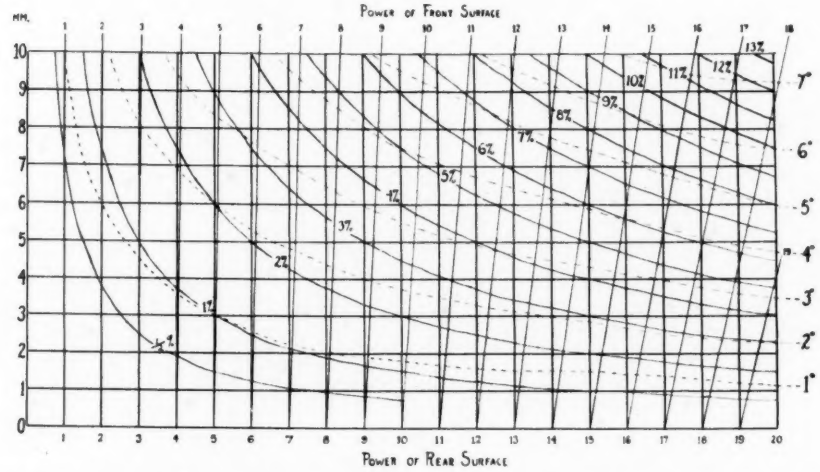


Fig. 5.—Nomogram of the magnifying power and peripheral prismatic effect of lenses of no dioptric power. The abscissas below represent the concavity of the rear surface, and those above the convexity of the front surface, in diopters. The ordinates represent the axial thickness of the lens in millimeters. The broken lines represent the family of lenses of equal peripheral prismatic power. The numbers associated with these lines give the total deviation in prism degrees with a rotation of the direction of gaze of 40 arc degrees. The solid curved lines represent the family of lenses of equal paraxial magnification.

lenses used for near and with power corrections combined with size corrections the relations become more complex. It becomes necessary, therefore, to compute the peripheral prismatic action of lenses of this type by actually tracing rays through them by the ordinary formulas of geometric optics.

In figure 5 I present a nomogram indicating the general characteristics of convexoconcave lenses of no dioptric power. The abscissas represent the concavity of the posterior surface in diopters. The

ordinates represent the axial thickness of the lens in millimeters. For a given depth of meniscus and a given axial thickness only one front surface curvature will result in zero dioptric power for the whole lens. This front dioptric power may be computed from the following formula:

$$\frac{1}{D_F} = \frac{1}{D_R} + T\,\frac{1}{\mu} \quad \ldots\ldots\ldots \quad (1)$$

in which D_F and D_R represent the front and rear dioptric power, respectively, T is the axial thickness (in meters) and μ the index of refraction of the glass.

From the chart one can immediately read, for instance, that a lens with a rear curve of -9 diopters and an axial thickness of 5 mm. must have a front curve of $+8.75$ diopters in order to have no axial dioptric power. The paraxial magnification is given by the formula:

$$M = \frac{T}{D_R \mu} \times 100 \quad \ldots\ldots\ldots \quad (2)$$

the magnification M being given in per cent. The lens just referred to shows a paraxial magnification of 3 per cent.

The peripheral prismatic action of these lenses has been computed for various angular rotations off axis of the direction of gaze on the assumption that the center of rotation of the eye lies 25 mm. behind the midpoint of the posterior surface of the lens. The broken lines in the chart represent the family of lenses of equal peripheral prismatic power. The numbers associated with these lines give the total deviation in prism degrees associated with a rotation of the direction of gaze of 40 arc degrees, that is, from 20 degrees above the optical axis to 20 degrees below the optical axis. If these lenses were perfect lenses, free from spherical aberration and distortion, the family of lenses having a total prismatic deviation of 4 prism degrees in 40 arc degrees of rotation would be identical with the family of lenses having 5 per cent paraxial magnification, but owing to the distortion produced by the lens the peripheral prismatic action of lenses of zero power is always a little less than is to be expected from the paraxial magnification for distant objects.

In patients who have an anisophoria either in all meridians or in a single meridian it is therefore possible to obtain a correction of the heterophoria by lenses of this type provided the degree of heterophoria is symmetrical about the axis of direct gaze and provided that the heterophoria increases uniformly as the gaze is directed away from the axis. It is plain, however, that if the patient initially suffers from anisophoria but has equal size of the images in the two eyes, one will produce aniseikonia in the correction of anisophoria. The converse also holds that if a patient initially has aniseikonia but no anisophoria the correction of the aniseikonia by a lens of this type will unquestionably

produce anisophoria, and since, in general, one must choose which of these two anomalies one wishes to correct, this particular aspect of the subject requires further discussion.

First it should be made plain that if initially the patient has neither anisophoria nor aniseikonia but only anisometropia, and one corrects his anisometropia by ordinary spectacle lenses, one will produce both aniseikonia and anisophoria as the result of the unequal magnification and unequal peripheral prismatic action of lenses of unequal dioptric power. If, by changing the base curve and thickness of one of the lenses one corrects the size difference between the two eyes, one will also at the same time and with equal accuracy (neglecting for the moment the unequal distortion of the two lenses) correct the patient's anisophoria. In this large group of cases, then, the correction of anisophoria and aniseikonia go hand in hand.

For the remaining cases, namely, those in which the patient is sufferring only from anisophoria and in whom the correcting lens produces aniseikonia, one has only the clinical results to prove that such a change may make for greater comfort. In my experience many patients of this group are relieved by this type of lens rather than made more uncomfortable. It may be mentioned, however, that considerable degrees of aniseikonia, if unaccompanied by anisophoria, can be cheerfully borne. For instance if, in reading at the usual reading distance, one displaces the book some 5 or 6 inches (13 to 15 cm.) to one side of the midline, the midpoint of the page will be from 5 to 6 per cent closer to one eye than to the other and the retinal image correspondingly larger in the abducted eye (fig. 6). Nevertheless, a simple trial of this kind will suffice to demonstrate that reading in such a position can be carried out without serious difficulty.

Dr. F. Walsh of the Wilmer Institute has recently studied a most interesting group of cases which also throw some light on this question. His study will be reported elsewhere but he has kindly permitted me to quote some of his findings. These patients all suffered from edema of the retina in the macular region of one eye associated with a very slight flat detachment of the retina in this region. All of those studied have spontaneously recovered. At the height of the trouble central vision was reduced to about 20/40, and there was a relative central scotoma. As recovery set in the patients noted a marked difference in the apparent size of objects seen with the two eyes, the difference amounting in some cases to as much as 20 or 30 per cent. The area of retinal edema extended over a roughly circular zone about the fovea, the radius of the circle being approximately one-half to two-thirds the distance from the fovea to the margin of the disk. The relative scotoma in the visual field correspondingly roughly filled the 10 degree circle on the tangent screen. It is to be presumed, therefore, that the difference in the size of the

retinal images was greatest in the fovea and decreased to zero at a distance of not more than 15 degrees from the center, and this was confirmed by rough tests. Within this zone, then, the patients suffered from all conceivable degrees of aniseikonia, but they had no disturbance in ocular motility, no anisophoria. These patients did not complain of any asthenopic symptoms, though they were somewhat troubled by the altered spatial appearance of things. Thus one patient stated that a golf ball appeared to him to be egg-shaped.

While the arguments which I have just advanced are sufficient to justify the use of lenses of this type by patients suffering from anisophoria, a final decision as to the relative importance of anisophoria and

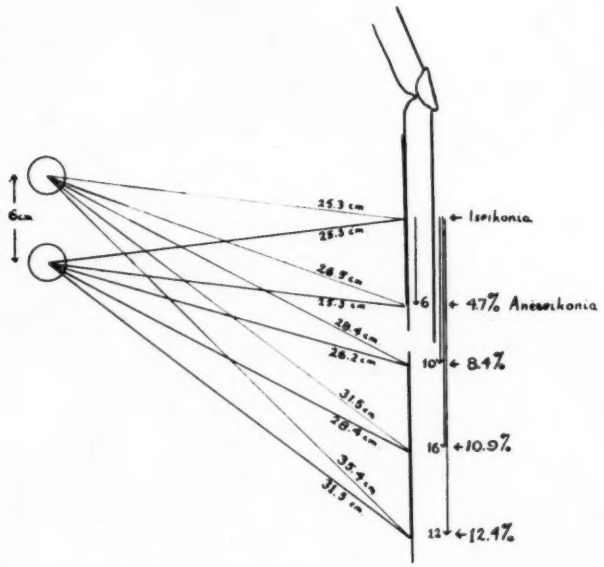


Fig. 6.—Effect of displacing reading matter to one side on the relative size of the ocular images.

aniseikonia in the production of symptoms can be reached only by making both types of measurement and alternate types of correction in a large group of cases. For the present one cannot even rule out the possibility that the two conditions may be intimately connected. A few illustrative cases will now be reported.

CASES IN WHICH ANISOPHORIA RESULTED FROM CORRECTION OF ANISOMETROPIA

CASE 1.—Miss B., aged 28, was first seen in 1931, when she complained of moderate asthenopia. The examination of her eyes revealed no organic disease. Refraction under a mydriatic gave the following results: In the right eye with +0.25 sph. vision was 20/20. In the left eye with +3.75 sph. vision was 20/20.

In view of the high degree of anisometropia and in view of the fact that she had never worn glasses, the following prescription was given her: right eye, plano; left eye, +1.75 sph. With these lenses she was comfortable, but in January 1935 she returned complaining of a recurrence of her earlier symptoms. Refraction under a mydriatic showed no change, and she was now given: right eye, plano; left eye, +3.50 sph. In one week she returned stating that she was unable to read with her new glasses; the letters became confused when she tried to read; she skipped lines and suffered from vertigo and from pains in her eyes. On measuring her lenses it was found that the optician had given her toric lenses as follows:

	Front Surface	Rear Surface	Thickness
Right eye	+6.00	—6.00	1.5 mm.
Left eye	+6.00	—2.50	3.0 mm.

With these lenses the results of tests of muscular balance were: on looking up 20 degrees, right hyperphoria of 2.5 diopters; on looking straight ahead, orthophoria; on looking down 20 degrees, left hyperphoria of 2.5 diopters. The optician was directed to substitute the following lens:

	Front Surface	Rear Surface	Thickness
Right eye	+9.75	—10.00	3.2 mm.
Left eye	No change		

With this change the results of tests of muscle balance were: on looking up 20 degrees, right hyperphoria of 0.5 diopter; on looking straight ahead, orthophoria; on looking down 20 degrees, left hyperphoria of 0.5 diopter.

When last seen she had worn these lenses for three months and had been entirely comfortable. It is to be noted that the reduction in the amount of heterophoria in this case was considerably greater than was to be anticipated from the peripheral prismatic power change made in her lens.

CASE 2.—Miss G., aged 62, was first seen in February 1935. She was a seamstress and had always had difficulty with her eyes, with tearing and burning of her right eye when she worked. Except for some slight vitreous opacities, her eyes were organically healthy. Refraction disclosed the following: In the right eye with 1.75 sph. —0.25 cyl., axis 45°, vision was 20/30. In the left eye with —3.50 sph. —0.50 cyl., axis 135°, vision was 20/30. There was a +3.00 addition for near. The glasses which she had been wearing were so nearly equal to this that the difference could not account for her symptoms. Examination of her heterophoria with her old glasses revealed: on looking up 20 degrees, left hyperphoria of 1 diopter; on looking straight ahead, orthophoria; on looking down 20 degrees, right hyperphoria of 1 diopter. The glasses which she had been wearing for near were ground with +7.00 sph. anterior surface and 1.5 mm. of central thickness. The following lenses were therefore prescribed:

	Front Surface	Rear Surface	Thickness
Right eye	+1.00 sph.	+0.25 cyl., axis 135°	1.5 mm.
Left eye	+7.00 sph.	—7.50 —0.50, axis 135°	2.0 mm.

With these lenses for the first time in many years she was able to read and sew with some comfort, but it was now found that she had between 0.75 and 1 diopter of left hyperphoria in all positions. The right lens was therefore modified to include a 0.75 diopter prism base up. With this change she had only a residual left hyperphoria of 0.25 diopter in all positions and was able to

read and sew with complete comfort. Distance glasses were now prescribed as follows:

	Front Surface	Rear Surface	Thickness
Right eye	−1.00 sph.	−0.75 sph. −0.25 cyl., axis 45° 1△ prism base up	1 mm.
Left eye	+5.50 sph.	−9.00 sph. −0.50 cyl., axis 135°	2 mm.

The patient is quite comfortable and, in spite of all that I can say to her, is much irritated with her previous ophthalmologist for not having helped her in this way.

CASE 3.—Mrs. B., aged 68, has been a patient of mine for many years. She has had senile cataracts and eight years ago had the right eye operated on and, six years ago, the left. Four years ago she was given glasses with results as follows: In the right eye with +11.50 sph. +2.00 cyl., axis 172°, vision was 20/20. In the left eye with +9.00 sph. +6.00 cyl., axis 20°, vision was 20/20. There was a +4.00 addition for near. With these glasses she was quite comfortable. Two years ago she was seized with headache, nausea, confused vision, vertigo and diplopia on looking upward and was found to have a paresis of the left superior rectus muscle. It was necessary at first to occlude the left eye in order to relieve her of her diplopia. Gradually the motility of the left eye returned, and six months ago she had, in looking straight ahead, only 2 diopters of right hyperphoria. A prism of this strength was placed in her right lens, and she reported that she could then see comfortably straight ahead but got dizzy in going up and down stairs and at street crossings, and that she still had to close her left eye in reading. Her glasses were found to have been ground as follows:

	Front Surface	Rear Surface	Thickness
Right eye	+13.25 sph.	−2.00 cyl., axis 82° + 2 △ prism base down	5.8 mm.
Left eye	+15.00 sph.	−6.00 cyl., axis 110°	6.0 mm.

With these lenses the results of tests of muscular balance were: on looking up 20 degrees, right hyperphoria of 2 diopters; on looking straight ahead, orthophoria; on looking down 20 degrees, left hyperphoria of 2 diopters. After a trial period during which she wore fronts of no dioptric power but of variable magnifying power over her glasses, the following lens was prescribed:

	Front Surface	Rear Surface	Thickness
Right eye	+16.75 sph.	−4.75 sph. + −2.00 cyl., axis 82° + 2△ prism base down	6 mm.
Left eye	No change		

With these glasses she was comfortable and her heterophoria was reduced to: on looking up 20 degrees, right hyperphoria of 0.5 diopters; on looking straight ahead, orthophoria; on looking down 20 degrees, left hyperphoria of 0.5 diopters. She now wears hook-on fronts over these distance glasses for reading.

This case seems to be of particular importance in the evaluation of the relative potency of aniseikonia and anisophoria in producing symptoms. Four years ago the patient had good binocular vision and was quite comfortable, though, owing to the difference in the refraction of her two eyes, the vertical meridian of the image of the left eye should have been larger than that of the right, while the horizontal image of the right eye should have been larger than that of the left. In the cor-

rection of the anisophoria which remained after recovery from the palsy of the left superior rectus muscle there was introduced an overall enlargement of the image for the right eye of about 3 per cent, decreasing her vertical aniseikonia but increasing her horizontal aniseikonia. Nevertheless, she is quite comfortable. It is to be remembered, however, that the amount of aniseikonia, like the amount of anisophoria, is not always strictly proportional to the magnifying power or the peripheral prismatic effect of the lenses which the patient is wearing.

CASE 4.—Mr. E., aged 25, was first seen in February 1935, when he complained that he was unable to read because his eyes filled with tears and burned when he attempted to do so. His vision, accommodation and fundi were all normal. Refraction gave results as follows: In the right eye with +0.25 sph. vision was 20/15. In the left eye with +0.25 sph. vision was 20/15. In testing his muscular balance with his gaze directed 20 degrees off axis in the various meridians the following complicated situation was found:

Tested at 10 Feet (3 Meters)

		Up		
Right	R.H. 1△	R.H. 2△	R.H. 3.5△	Left
	R.H. 1△	R.H. 2△	R.H. 3.5△	
	R.H. 1.5△	R.H. 1△	R.H. 3.5△	
		Down		

Tested at 1 Foot (33 Cm.)

		Up		
Right	R.H. 3△	R.H. 3△	R.H. 5△	Left
	R.H. 2△	R.H. 6△	R.H. 6.5△	
	R.H. 1.5△	R.H. 3△	R.H. 8△	
		Down		

In the results at 10 feet it is plain that in traveling along the meridian of 135 degrees his left eye made a movement with a vertical component 2.5 prism degrees larger than the right. For oblique movements to have such a difference in vertical component the movement of the left eye in the meridian of 135 degrees must be 3.5 prism degrees larger than that of the right. From such considerations one reaches the conclusions embraced in figure 7.

The interpretation of these findings is a matter of some interest. Since there is overall a considerable amount of right hyperphoria there must be a relative weakness of one of the depressors of the right eye or of one of the elevators of the left eye. Since the disturbance increases on looking toward the left the weak muscle must be either the superior oblique muscle of the right eye or the superior rectus muscle of the left, and since the disturbance increases in convergence it must be the right superior oblique muscle that is primarily affected. This accounts for the left macrophoria in the meridian of 135. By analogy one concludes that there is also a weakness of the left superior oblique muscle of lesser degree. Finally, from the fact that the heterophoria did not disappear on upward gaze it is to be presumed that there is also some spasm or contracture of the inferior oblique muscles. It is evident that the patient requires a magnification of the meridian of 45 for his right eye and of the meridian of 135 for his left eye. Since the full correction for near work could not be ground in an ordinary spectacle lens, the following partial correction was prescribed, and the patient was directed to wear the glasses constantly for a week.

	Front Surface	Rear Surface	Thickness
Right eye	+9.75 cyl., axis 135°	−10.00 cyl., axis 135°	3 mm.
Left eye	+9.75 cyl., axis 45°	−10.00 cyl., axis 45°	3 mm.

The patient returned in a week stating that his eyes were comfortable and that he could read without difficulty, but that everything looked stream-lined to him,

and that his glasses looked stream-lined to his friends, and "couldn't we try something else."

With these glasses his heterophoria was as shown in the accompanying diagrams.

	Tested at 10 Feet			
Right	R.H. 0.5△	R.H. 0.5△	R.H. 0.5△	Left
	R.H. 0.75△	R.H. 1△	R.H. 1△	
	R.H. 1△	R.H. 1△	R.H. 1.5△	

	Tested at 1 Foot			
Right	R.H. 4△	R.H. 4.5△	R.H. 5△	Left
	R.H. 3△	R.H. 5△	R.H. 5△	
	R.H. 2△	R.H. 5△	R.H. 6△	

It is to be noted that the reduction in the heterophoria, especially for near, is much greater than was to have been expected from the peripheral prismatic action of the lenses, and that there is no optical reason for diminution in heterophoria in the vertical meridian. As with case 1, I have no explanation for this phenomenon. Since the overall right hyperphoria still persisted with these glasses, and since it was clear that the patient was able to tolerate a considerable degree of uncorrected anisophoria, it was thought worth while to try

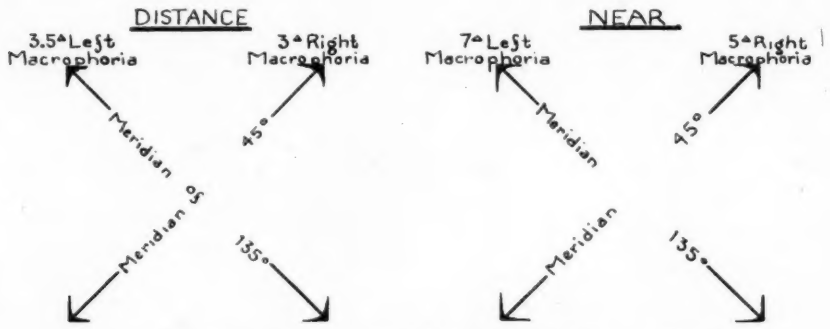


Fig. 7.—Diagrammatic representation of the anisophoria in case 4.

the correction of the right hyperphoria alone and leave the anisophoria uncorrected. He was therefore given: right eye, plano +1 Δ prism base down; left eye, plano.

These lenses were not so comfortable for reading as the previous ones, but the world looked more natural and his friends no longer teased him. Furthermore, the lenses afforded him some relief from his former symptoms, and he preferred the residual physical discomfort with these lenses to the esthetic discomfort with the former pair.

DISCUSSION OF ANISOPHORIA DUE TO MOTOR ANOMALIES

It may be seen from the last two cases that isolated weakness of one extra-ocular muscle with contracture of the opposing muscle can be compensated for with the type of lens that I have suggested, or in some cases with ordinary prisms, and that there are some cases of bilateral symmetrical lesions that can also be dealt with. It seems worth while, therefore, to enumerate the various permutations and combinations which may occur and their differential diagnosis. This is done in the appended table.

The list of possibilities given in the table is not necessarily exhaustive. However, it does cover all the types I have discovered so far. It will be seen that isolated paresis of one vertical rotator (1, 3, 5 and 7 in the table) without contracture of the antagonist yields a form of anisophoria that can be aided with prisms plus either overall or meridional magnification on the nonparetic eye. Perfect correction cannot, however, be obtained, though, as in case 3, symptomatic cure can be attained. Isolated paresis of a single vertical rotator with contracture of the antagonist (2, 4, 6 and 8) may be perfectly corrected by combining prisms with meridional magnification, provided the contracture exactly balances the paresis and yields no variation in the heterophoria in the domain of mutual action of these muscles. For such a variation an overall or meridional magnification for one eye only combined with use of prisms may, as in the previous group, yield a closer approximation.

Bilateral symmetrical weakness of one vertical rotator of each eye with contracture of the antagonist (13, 14, 15 and 16) is by no means rare. Perhaps it is more nearly correct to regard such a combination as a muscular imbalance between the oblique muscles and the vertical recti rather than as paresis and contractures. Most of the patients with this condition are without symptoms, and the one patient with the condition whose muscular imbalance I have corrected (case 4) was relieved of his symptoms but rejected the correction for esthetic reasons.

Paresis of a vertical rotator of one eye with spasm of the antagonist to the corresponding muscle of the other eye (9, 10, 11 and 12) undoubtedly occurs and, in some instances, is responsible for the production of symptoms, but I have found no way in which anomalies of this type can be systematically corrected. Usually the paresis and spasm do not exactly balance each other, so that the anomaly becomes clearly manifest only after correction of the overall heterophoria with prisms. Bilateral symmetrical disturbances of this type (17, 18, 19 and 20) are frequently encountered but do not, as a rule, cause symptoms. Again it might be more correct to consider this type as an imbalance between the oblique muscles and the vertical recti. As a matter of fact, the type of anisophoria which is present is precisely the same as that physiologic anisophoria which, as I have already pointed out, is normally associated with altered degrees of convergence. Thus, if one places before a normal patient moderately strong concave spherical lenses and forces him to accommodate during the test for heterophoria, one finds precisely the disturbance illustrated in 18 and 19. Conversely, if one provides a presbyope with reading glasses and tests his heterophoria at the near range without allowing his eyes to accommodate or converge, one finds the disturbance illustrated in 17 and 20. It is no wonder, therefore, that patients who have gone for many years with an uncorrected spherical

Combinations of Muscle Imbalance and the Results of Corrective Measures

Anomaly	Field of Heterophoria	Result of Correction with Prism	Result of Correction with Prism Plus Meridional Magnification	Result of Correction with Prism Plus Meridional Magnification Plus Overall Magnification	Comment
1. Paresis of right superior rectus	L{L+ L++ L+++ / 0 L+ L++ / 0 0 0}R	L{0 L+ L++ / R+ 0 L+ / R+ R+ R+}R	L{0 L+ L+ / R± 0 L±}R / 0 R± R+}	L{0 0 L± / R± 0 L± / L± 0 R±}R	Fairly good correction can be obtained with prism plus overall magnification without meridional magnification as in case 3
2. Paresis of right superior rectus plus contracture of right inferior rectus	{L+ L++ L+++ / L+ L++ L+++ / L+ L++ L+++}	{R+ 0 L+ / R+ 0 L+ / R+ 0 L+}	{0 0 0 / 0 0 0 / 0 0 0}		Prisms alone are fairly satisfactory; if the residual heterophoria after prismatic correction is perfectly symmetrical, complete correction by magnifying opposite oblique meridians in the two eyes is possible
3. Paresis of right inferior rectus	{0 0 0 / 0 R+ R++ / R+ R++ R+++}	Analogous to 1			
4. Paresis of right inferior rectus plus contracture of right superior rectus	{R+ R++ R+++ / R+ R++ R+++ / R+ R++ R+++}	Analogous to 2			
5. Paresis of right superior oblique	{0 0 0 / R+ 0 0 / R+++ R+ 0}		{0 L± L+ / R± 0 L± / R+ R± 0}		Heterophoria increases with convergence; use meridional correction for near work only
6. Paresis of right superior oblique plus contracture of right inferior oblique	{R+++ R++ 0 / R+++ R++ 0 / R+++ R++ 0}	Analogous to 2, but heterophoria increases with convergence			Prisms alone are fairly satisfactory; stronger prisms may be required for reading
7. Paresis of right inferior oblique	{L+++ L+ 0 / L+ 0 0 / 0 0 0}	Analogous to 5			
8. Paresis of right inferior oblique plus contracture of right superior oblique	{L+++ L+ 0 / L+++ L+ 0 / L+++ L+ 0}	Analogous to 6			

9. Paresis of right superior rectus plus spasm of left superior oblique	0 0 0	0 0 0	L+ 0 R+	Right hyperphoria on downward gaze increases with convergence	Prism may be required for reading; no general correction is possible
10. Paresis of right inferior rectus plus spasm of left inferior oblique	Same as 9			Left hyperphoria on upward gaze increases with convergence	No correction is possible
11. Paresis of right superior oblique plus spasm of left inferior rectus	L+ 0 R+	0 0 0	0 0 0	Left hyperphoria on upward gaze increases with convergence and with tilting of head	No correction is possible
12. Paresis of right inferior oblique plus spasm of left superior rectus	Same as 11			Right hyperphoria on downward gaze increases with convergence	Prism may be required for reading; no general correction is possible
				Result with Bilateral Meridional Magnification	
13. Bilateral paresis of superior obliques with contracture of inferior obliques	R+ R+ R+	0 0 0	L+ L+ L+	0 0 0 0 0 0 0 0 0 This condition is similar to 2, but heterophoria increases with convergence and lacks initial overall heterophoria	This requires meridional correction on both eyes
14. Bilateral paresis of inferior obliques with contracture of superior obliques	L+ L+ L+	0 0 0	R+ R+ R+	0 0 0 0 0 0 0 0 0 Heterophoria increases with convergence	This required meridional correction on both eyes
15. Bilateral paresis of superior recti with contracture of inferior recti	R+ R+ R+	0 0 0	L+ L+ L+	Identical with 13, but heterophoria fails to increase with convergence	This required meridional correction on both eyes
16. Bilateral paresis of inferior recti with contracture of superior recti	L+ L+ L+	0 0 0	R+ R+ R+	Identical with 14, but heterophoria fails to increase with convergence	This required meridional correction on both eyes
17. Bilateral paresis of superior recti with spasm of superior obliques	R+ 0 L+	0 0 0	L+ 0 R+	Heterophoria on downward gaze should increase with convergence	No correction is possible
18. Bilateral paresis of inferior recti with spasm of inferior obliques	L+ 0 R+	0 0 0	R+ 0 L+	Heterophoria on upward gaze should increase with convergence	No correction is possible
19. Bilateral paresis of superior obliques with spasm of superior recti	L+ 0 R+	0 0 0	R+ 0 L+	Heterophoria on downward gaze should increase with convergence	No correction is possible
20. Bilateral paresis of inferior obliques with spasm of inferior recti	R+ 0 L+	0 0 0	L+ 0 R+	Heterophoria on upward gaze should increase with convergence	No correction is possible

ametropia or with a marked horizontal muscular imbalance may show varying forms of this type of anisophoria.

SUMMARY AND CONCLUSIONS

Anisophoria is defined as that form of heterophoria in which the degree of muscular imbalance varies with the direction of gaze. Methods for the detection and measurement of anisophoria are outlined. Anisophoria may be produced by the correction of anisometropia with ordinary spectacle lenses. In these cases it is due to the unequal prismatic action of the peripheral portions of the lenses. Anisophoria may also result from the unequal action of the extra-ocular muscles. In such cases it is usually associated with some heterophoria on direct gaze. The various types of anisophoria produced by the more common forms of rotational inequality of the eyes are discussed. The physiologic rotational inequalities of the eyes are also discussed, and some relations between these and the pathologic forms of anisophoria are pointed out. Three methods for the correction of anisophoria are described: the use of segments of prisms; the displacement of reading matter toward the side of the eye which has the greater rotational amplitude, and the magnification of the image for the eye which has the greater rotational amplitude. A rough method for the computation of such magnifying lenses is outlined, and the need in some cases for meridional magnification rather than overall magnification is pointed out. The relative merits and limitations of these three methods for the correction of anisophoria are discussed, and a few illustrative cases are reported showing the third type of correction.

ABSTRACT OF DISCUSSION

DR. WALTER B. LANCASTER, Boston: I am inclined to think that Dr. Friedenwald has overstated his case, but if that is true, time will correct the overstatement, and it has the merit of giving emphasis to his claims.

There can be no doubt that anisophoria exists. It throws a burden on the adjusting mechanisms of binocular vision. The most frequent causes of eye strain involve the accommodative mechanism. In some way adjustments can be made for differences in size and shape of the ocular images—aniseikonia. The ophthalmologist's job is to relieve eye strain by treating one or more of the three defects of adjustment due to refractive errors, phorias and aniseikonia. Dr. Friedenwald has emphasized the importance of a heretofore neglected type of phoria, anisophoria. Is this as important as he states, and does it vitiate the claims for aniseikonia as he implies?

Of the existence of anisophoria and of its relative frequency there can be no doubt. The author is not ready to define the best way to measure it. He chooses to measure the phoria at 20 degrees from the primary position because, if there is anisophoria, it is much larger at 20 degrees from the primary position than at 5 degrees.

I feel that Dr. Friedenwald in his classification of anisophoria into types has overstressed the individual muscle and its antagonists and underrated the

coordinate action of the whole mechanism with deviations approximating concomitant deviations rather than a picture seen in fresh paresis, which is the type of disability of the individual muscle. Moreover, it seems more probable that defects in the adjustments of the neuromuscular mechanism would predominate over breakdown or paresis of a single muscle.

Now, it is obvious that the movements inside the 5 degree radius are so enormously frequent and important that the neuromuscular mechanism is more perfect, works more smoothly, for these than for movements outside that range. When one approaches the limits of rotation the tasks of the fusion reflex and the fixation reflex become so great that, as the jerky, nystagmus-like movements show, they are at the breaking point.

I believe, therefore, that only after careful study and accumulation of sufficient corroborative data should one accept the readings at 20 degrees from the primary position as applicable at 5 degrees, and that one should not be too ready to classify phorias as due to single, individual muscles.

Passing from the measurement of anisophoria to its correction, one invades the sphere of activity of Professor Ames and his staff, who have done so much on the theory and design of lenses to produce magnification without changing the focal power. These are covered by patents held by Dartmouth College.

Dr. Friedenwald has not made it plain that the design and especially the manufacture of these lenses present great difficulties.

Since this type of lens is to correct anisophoria, and the same type of lens is used to correct aniseikonia, it is obvious that these two should be considered together. No intelligent and final discussion of either is possible without considering both.

Dr. Friedenwald has not given sufficient emphasis to the work of the Dartmouth investigators. One of the most consistent of their findings is the effect of aniseikonia and its correction on heterophoria and heterotropia. This is well known to Dr. Friedenwald.

An outstanding claim of those investigating aniseikonia is that in many cases its correction corrects heterophoria and even heterotropia. Striking instances are described by Bielschowsky. This is accomplished not directly by the optical effect of the lenses, but indirectly by removing the cause, to wit, the aniseikonia.

Dr. Friedenwald over and over again in his paper assumes that aniseikonia is present or absent, as his argument may require, without attempting to determine the fact by measurement. This is the most weighty criticism of his paper. It is to be deplored, because the conception which is his main thesis is probably a very valuable contribution to ophthalmology.

Dr. F. H. Adler, Philadelphia: It may be the habit of some ophthalmologists to measure the deviations of the eyes from their normal relationship one to the other at different positions in space by tilting the head on the neck and on the trunk instead of changing the position of the fixation light. That has been the practice of Landolt in measuring heterotropia in different positions of gaze, and it is the practice in some clinics in this country. Instead of changing the position of the fixation light, the patient's head is tilted on the chest or the chin is elevated, depressed or moved from side to side.

When heterophoria is thus measured the position of the eyes is determined with one factor, one source of tone, removed, that is the source of tone due to fusional impulses. The other sources of tone, which go to the extra-ocular muscles, are still present.

One knows that these other sources are (1) the reflexes coming from the otoliths and hence called labyrinthine reflexes, and (2) the impulses which arise from the muscles of the neck, known as neck reflexes.

If one wishes to determine heterophoria in different positions of gaze and changes the position of the head in respect to the neck, or by tilting stimulates the otolith apparatus, the determinations will be erroneous and not what one sees. Therefore, if one is to determine anisophoria in the future, it will be necessary, I believe, to employ an apparatus that changes the position of the fixation light.

Dr. Conrad Berens, New York: I should like to know whether it is a simple procedure to measure small deviations accurately with prisms at 12 inches (30.5 cm.) and how these studies are made. My associates and I have found many variable factors and numerous difficulties in this type of measurement.

I should also like to know how many and what types of cases were studied in order to determine normal or physiologic anisophoria and whether these patients had aniseikonia.

Dr. Friedenwald states that in pathologic cases overall heterophoria must be corrected to elicit anisophoria. How far from the point of fixation were these determinations made? In hyperphoria, far out in the periphery in the "four corners," vertical deviations, which vary if the lateral deviation is corrected, can usually be detected.

Dr. Friedenwald mentions severe symptoms from 0.5 diopter of hyperphoria found only in the extremes of gaze. In my experience hyperphoria of this degree, even when occurring in the primary position, has produced few symptoms. However, aniseikonic patients with low degrees of hyperphoria have apparently been relieved by their iseikonic correction. For example, an ophthalmologist whose symptoms were not relieved by prismatic lenses, orthoptic treatment or surgical operation was later relieved by wearing a weak correction for aniseikonia. Iseikonic correction at times seems to change the heterophoria.

It has also often been found practical to have the patient in using the eyes select the position in which his deviation varies least. A book rest above the horizontal plane is useful, and frosting or otherwise excluding part of the field is valuable.

I have found orthoptic training valuable in increasing a patient's tolerance to variable hyperphoria. This may be attributed to improved tolerance to aniseikonia as well as to increased amplitude of fusion and the like.

The department of research in physiologic optics at Dartmouth Medical School formerly varied the base curves of lenses, but this is now done only after careful tests for aniseikonia. Dr. Friedenwald points out that aniseikonia is produced by this type of correction and states that the refraction is also changed, hence the need for the ophthalmo-eikonometer and other methods of studying aniseikonia and the simultaneous study of ametropia, heterophoria and aniseikonia. It is not clear to me whether Dr. Friedenwald has made these simultaneous studies.

According to Dr. Friedenwald, anisophoria causes more symptoms than aniseikonia. Although I have apparently observed fair fusion in and near the primary position after unilateral extraction of cataract, the patients were not studied for aniseikonia. In the department of research in physiologic optics at Dartmouth Medical School continuous binocular vision has not been found when the difference in size was greater than 4 per cent. The ability to overcome great differences in size possibly may be limited to the foveal region.

Many cases will have to be studied before the relative importance of anisophoria and aniseikonia can be evaluated finally.

I hope to use Dr. Friedenwald's exact method in conjunction with the present methods of studying heterophoria in relation to aniseikonia.

DR. JONAS S. FRIEDENWALD, Baltimore: In regard to Dr. Lancaster's question as to whether I have overstated the case, I should like to make it clear that, while anisophoria is very common and one can discover it in from 20 to 30 per cent of cases of error in refraction, the number of cases in which it seems to produce symptoms is limited.

What is the relation of this phenomenon to aniseikonia? My study was originally undertaken for the following reason: Aniseikonia is tested by the so-called ophthalmo-ikonometer. It is apparent that if a patient has anisophoria he will erroneously seem to have aniseikonia when tested with the ikonometer. My investigation has shown that anisophoria is common and that in some instances it produces symptoms similar to those which are attributed to aniseikonia. There is no way to guess what proportion of the therapeutic success attributed to the use of size lenses is due to the correction of aniseikonia and what portion is due to the correction of anisophoria. In the famous case reported by Professor Bielschowsky, for instance, the patient suffered from complicated rotational disparity between the two eyes. Size lenses were prescribed for him which produced an extended field of binocular single vision. The maximal supposed size difference was found in the meridian of action of a recently tenotomized muscle. Owing to his faulty perception he has never been tested with the horopter or the tilting field.

In some of the cases that I have reported the expected prismatic action of the lenses which I prescribed was much less than the actually achieved reduction of the anisophoria.

As regards most common types of anisophoria, Dr. Lancaster is correct in stating that isolated pareses of individual muscles are much rarer than disparities in coordination of muscle groups. By and large I have found the most frequent disparity to be that between the vertical recti of one eye and the oblique muscles of the opposite eye. This, of course, emphasizes the importance of Dr. Adler's statement that the test should be made with the head in the erect position, moving the test object rather than the head.

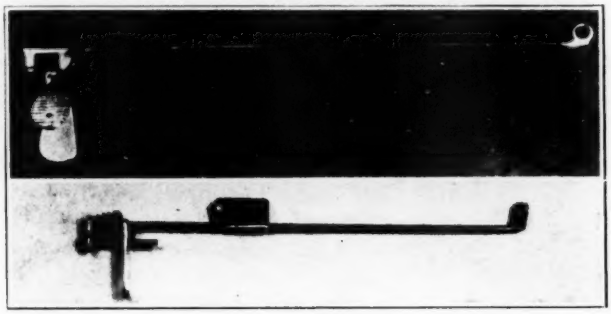
Clinical Notes

A NEW FORCEPS FOR REMOVAL OF LEAD SHOT FROM THE VITREOUS

HARVEY E. THORPE, M.D., PITTSBURGH

The removal of lead shot from the vitreous has always presented a difficult problem for the ophthalmologist. The use of roentgen rays has facilitated the localization of the opaque foreign body.

A forceps is described in this paper with which it is possible to remove a lead shot from the vitreous with a minimum of manipulation. This miniature forceps consists of a tubular shaft with a free-moving center rod. These are attached at one end to two ring-shaped blades,



Forceps for the removal of lead shot from the vitreous chamber.

which act as the blades of the forceps. The blades surround the lead ball and hold it by means of spring action, which is applied at the manipulating end of the forceps. This manipulating end consists of two handles, pressure on which opens the blades of the forceps. The diameter of the tubular shaft is 1 mm. The forceps may be inserted into the eye through a 5 mm. linear incision and operated with a minimum of manipulation while the forceps is in the wound.

Observation of the action of the forceps may be through the pupil if the lens is clear or through the ocular endoscope.[1] The forceps [2] may also be used with the aid of the two plane fluoroscope or with a transilluminator applied to the opposite side of the globe, which casts a shadow of the shot and the forceps.

Read before the Section on Ophthalmology at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 13, 1935.

1. Thorpe, Harvey E.: Ocular Endoscope, Tr. Sect. Ophth., A. M. A., 1934, p. 290; Ocular Endoscope, Tr. Am. Acad. Ophth., 1934, p. 422.

2. This instrument was made by the American Cystoscope Makers, Inc., New York.

WHITE RING IN THE CORNEA

YASUO UYAMA, OSAKA, JAPAN

EDITED BY WALTER F. DUGGAN, M.D., NEW YORK

Reports concerning a white ring in the cornea have recently appeared in the literature. The original communication was made by Coats [1] in 1912. Muramatsu,[2] who also reported five new cases, recently reviewed the literature to date.

While this particular corneal lesion is not rare, it is seldom observed clinically because (1) there is so little subjective disturbance that the patient does not seek medical advice, and (2) the lesion is so tiny that it is probably often overlooked. Hence, little is known of the nature or development of this lesion.

The necessity of removing a foreign body from a cornea in which a typical white ring was present adjacent to the foreign body afforded the opportunity of removing a portion of the white ring at the same time.

REPORT OF CASE

K. K., a woman aged 42, was originally seen because of a bilateral dimming of vision due to syphilitic retinitis. The vision was 0.9 in each eye, and, except for the retinitis the only other pathologic change present was a white ring in the right cornea. The Wassermann reaction of the blood was strongly positive. The urine contained no albumin or dextrose, and the family history was irrelevant. The patient had had four children, one of whom had died of meningitis. Under anti-syphilitic treatment the retinitis subsided, and the vision improved.

The white ring, which was situated just below and nasal to the center of the right cornea, was elliptic, with an inner diameter of about 0.5 mm.

Slit-lamp examination showed, as in Muramatsu's first case, a small, ring-shaped, chalky white opacity; the ring was incomplete temporally. The inner and outer edges of the dense white opacity were fringed by fine powdery opacities which formed a complete circle (fig. 1). The ring itself was superficially situated, but the overlying epithelium was normal and did not stain with fluorescein.

On removing the ring with a needle (at the time that a foreign body was removed from another part of the cornea) it was found that the powdery opacities were situated more deeply than the ring itself. The ring was placed on a glass slide, fixed with formaldehyde and stained with sudan III.

On histologic examination (fig. 2) a small mass of orange-yellow granules was seen in the superficial layer of the cornea along its surface. A nuclear stain was not used because of the small amount of material available; so, the relation of the granules to the corneal epithelium and Bowman's membrane could not be determined. However, in accordance with the result of the slit-lamp examination, it seems certain that the granules lie in the upper part of the cornea, very close to

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From the Osaka Imperial University.

1. Coats, G.: Two Cases Showing a Small, Superficial, Opaque, White Ring in the Cornea, Tr. Ophth. Soc. U. Kingdom **32**:52, 1912.

2. Muramatsu, N.: Five Cases of White Ring in the Cornea, Chuo-Ganka-Iho **27**:32, 1935.

the epithelium. Moreover, since the material composing the white ring stains with sudan III, it seems certain that it is composed of fat.

Muramatsu, who investigated his third case histologically, found that the epithelium, though thin, was intact and level over the opacity. Under Bowman's membrane there was a homogeneous, purple-staining

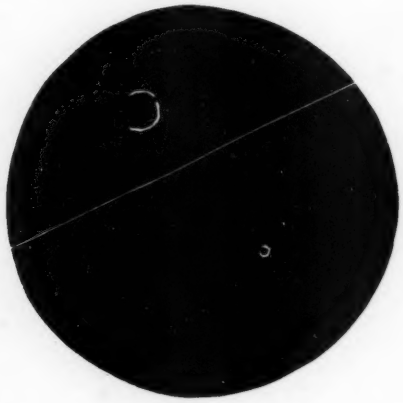

Fig. 1.—The right cornea of the patient. The lower portion shows the white ring as seen by the unaided eye. The upper portion shows details of the white ring as seen with the slit lamp.

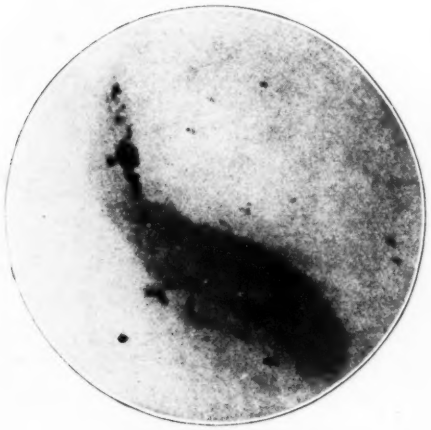

Fig. 2.—Portion of the white ring of the cornea stained with sudan III. The orange-yellow granules are located chiefly at the upper (left) end of the specimen.

substance which threw Bowman's membrane into folds and which was apparently situated between Bowman's membrane and the substantia propria of the cornea.

It would seem that the homogeneous, purple-staining substance found by Muramatsu is identical with the granules which took the sudan III stain in my case.

Various views are held as to the origin of the white ring of the cornea. Coats [1] and Mayou [3] assumed a congenital origin; Ballantyne [4] ascribed it to changes in the globe, while Vogt [5] and Gallemaerts [6] expressed the opinion that it is due to trauma.

In my opinion a white ring of the cornea is due to the deposition of fat on a small scar under the corneal epithelium. It is assumed that the scar developed following a foreign body of the cornea which had penetrated to the substantia propria of the cornea and had then fallen off with regeneration of the corneal epithelium over the site of injury.

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3. Mayou, M. S.: White Ring in the Cornea, Brit. J. Ophth. **17**:342, 1933.

4. Ballantyne, A. J.: White Rings in the Cornea, Brit. J. Ophth. **17**:336, 1933.

5. Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, ed. 2, Berlin, Julius Springer, 1930.

6. Gallemaerts, E.: Examen microscopique des affections de la cornée au moyen de la lampe à fente, Paris, Masson & Cie, 1926.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

THE SCHEMATIC EYE

ALFRED COWAN, M.D.

PHILADELPHIA

The refractive surfaces and media of the eye consist of the anterior convex surface of the cornea, the posterior concave surface of the cornea, the aqueous contained in the meniscus-shaped anterior chamber, the elastic biconvex crystalline lens and the vitreous.

To follow the passage of light through such a system is a tedious and complicated procedure, and in order to facilitate the study of the optical imagery it is advantageous to construct what may be considered a diagrammatic representation of an ideal, normal eye. Such an optical system is called a schematic eye. Schematic eyes are based on a careful analysis of the dioptric systems of a number of emmetropic eyes. The curvatures and indexes of refraction of the various refracting units and their distances, one from the other, of each eye are measured with extreme exactitude and then averaged, and the result is considered by the investigator to be the standard or ideal average eye.

In every schematic eye the refracting surfaces are centered on a line passing through the middle of the system, which is called the principal axis. The points where the principal axis cuts the system in front and behind are called the poles. The posterior pole passes through the retina in a point between the fovea and the entrance of the optic nerve, and the anterior pole cuts the vertex of the anterior corneal curve. Also, it is assumed that the object is infinitely small and that the eye is provided with an infinitely small aperture or pupil, so that the pencil of light which enters the eye is infinitely small; that is, all the rays are supposed to lie very close to the principal axis, in what is called the gaussian space.

To make calculations easier for practical purposes and for conditions in which results need not be exact, the optical constants may be carried out to fewer decimal places, and certain small values may be considered negligible. This is called a simplified schematic eye. For rough calculations a reduced eye, one with almost round numbers, may be used.

Probably the most generally accepted of the numerous schematic eyes which have been constructed since that of Listing is that of Gullstrand. The optical constants of Gullstrand's exact schematic eye and

of his simplified schematic eye will be discussed in the order given in the table. The refracting powers are expressed in diopters and the linear measurements in millimeters.

Gullstrand's Schematic Eye *

	Exact		Simplified	
	Accommodation Relaxed	Maximum Accommodation	Accommodation Relaxed	Maximum Accommodation
Index of refraction				
Cornea	1.376	1.376		
Aqueous and vitreous	1.336	1.336	1.336	1.336
Lens	1.386	1.386	1.413	1.424
Equivalent core lens	1.406	1.406		
Position				
Anterior surface cornea	0	0	0	0
Posterior surface cornea	0.5	0.5		
Anterior surface lens	3.6	3.2		
Anterior surface equivalent core lens	4.146	3.8725		
Posterior surface equivalent core lens	6.565	6.5275		
Posterior surface lens	7.2	7.2		
Optical center lens	.....	.....	5.85	5.2
Radius curvature				
Anterior surface cornea	7.7	7.7		
Posterior surface cornea	6.8	6.8		
Equivalent surface cornea	.....	.....	7.8	7.8
Anterior surface lens	10.0	5.33	10.0	5.33
Anterior surface equivalent core lens	7.911	2.655		
Posterior surface equivalent core lens	−5.76	−2.655		
Posterior surface lens	−6.0	−5.33	−6.0	−5.33
Refracting power				
Anterior surface cornea	48.83	48.83		
Posterior surface cornea	−5.88	−5.88		
Equivalent surface cornea	.....	.....	43.08	43.08
Anterior surface lens	5.0	9.375	7.7	16.5
Core lens	5.985	14.96		
Posterior surface lens	8.33	9.375	12.833	16.5
Corneal system				
Refracting power	43.05	43.05	43.08	43.08
Position of first principal point	−0.0496	−0.0496	0	0
Position of second principal point	−0.0506	−0.0506	0	0
First focal length	−23.227	−23.227	−23.214	−23.214
Second focal length	31.031	31.031	31.014	31.014
Lens system				
Refracting power	19.11	33.06	20.53	33.0
Position of first principal point	5.678	5.145	5.85	5.2
Position of second principal point	5.808	5.225	5.85	5.2
Focal length	69.908	40.416	65.065	40.485
Complete optical system of eye				
Refracting power	58.64	70.57	59.74	70.54
Position of first principal point	1.348	1.772	1.505	1.821
Position of second principal point	1.602	2.086	1.631	2.025
Position of first focal point	−15.707	−12.397	−15.235	−12.355
Position of second focal point	24.387	21.016	23.996	20.963
First focal length	−17.055	−14.169	−16.740	−14.176
Second focal length	22.785	18.930	22.365	18.398
Position of fovea centralis	24.0	24.0	24.0	24.0
Axial refraction	−1.0	−9.6	0	−9.7
Position of near point	.....	−102.3	.....	−100.8

* The refracting powers are expressed in diopters and the linear measurements in millimeters.

The index of refraction of the cornea is taken as 1.376 after Matthiessen, and that of the vitreous and aqueous, as 1.336, after Helmholtz. The dioptric power of the eye could hardly be lowered, even if water were substituted for normal aqueous. On the other hand, if the index

of refraction of the aqueous were increased to 1.36, that of pure albumin, the dioptric power of the eye would be increased by only about 3 diopters.

The anterior surface of the cornea was formerly represented as ellipsoid; but it can be concluded from the results of numerous investigations that, in general, with a pupil of medium size the assumption of a spherical shape for the utilized part of the cornea is provisionally the best approximation. According to Gullstrand, the central spherical portion of the cornea extends about 4 mm. horizontally and somewhat less vertically. It is decentered outward and slightly downward. The pupil is also decentered outward and slightly downward, corresponding very nearly with the optical zone of the cornea, so that in most typically regular eyes the latter can be considered as approximately centered with respect to the pupil. Accordingly, it is best to take the normal to the cornea, which goes through the center of the pupil, as being the optical axis of the eye. This is given as 0 in the table, because it is from this point on the principal axis that all distances are measured, those in front having the negative sign. The radii of curvature are given the positive sign when convex and the negative sign when concave from before backward.

The position of the posterior surface of the cornea is given as 0.5 mm.; that is, the cornea is 0.5 mm. in thickness at the anterior pole, as proposed by Blix. This is less than the thickness usually given in the textbooks, but it is probably more nearly accurate for the cornea of the living eye. Tscherning, who was the first to consider the posterior surface of the cornea in a schematic eye, gave 1.15 mm. for the thickness. This value is unquestionably too high.

The distance from the anterior surface of the lens to the anterior surface of the cornea, when the accommodation is relaxed, is in agreement with that of Helmholtz and other investigators. Tscherning gave this distance as 3.54 mm., a very fine distinction for a mean value, when admitted sources of error must be taken into account.

Although it is well known that the index of refraction of the substance of the lens increases from the surface toward the center, most schematic eyes are based on the assumption that the crystalline lens is biconvex, of an isotropic medium having the same index of refraction throughout. Gullstrand showed that it is not true that the substance of the lens is composed of a medium of continuously variable index of refraction. In the second and third decades of life it can be seen with the slit lamp and corneal microscope that there are distinct zones with separating surfaces. The surfaces separating the zones of different refractive index are called surfaces of discontinuity, for the reason that they separate different media of variable index of refraction. More-

over, the index of refraction changes with every change of the shape of the lens and cannot be calculated at all from the index of refraction at the center of the lens and in the outside cortical layer. This, together with the postmortem variations, accounts for the great difference in values as given by different authorities. There is no way, therefore, of calculating the index of refraction of the crystalline lens except in the living eye; and the only method remaining is to calculate it from the loss of refraction that the eye suffers when the lens is extracted.

Gullstrand chose a point approximately 10 cm. distant for the near point of his accommodating eye and retained Helmholtz' figure of 0.4 mm. for the forward displacement of the anterior surface of the lens during maximum accommodation. Gullstrand assumed, for his calculations, that the lens consists of an anterior surface lens and a posterior surface lens, each having an index of refraction of 1.386, and an equivalent core lens, which is 2.4146 mm. in thickness along the axis, with an index of refraction of 1.406. During accommodation the anterior surface of the core lens comes forward 0.2735 mm. The posterior surface also moves forward, but only 0.0375 mm., so that the thickness increases from 2.419 to 2.655 mm.

The position of the posterior surface of the lens, 7.2 mm. behind the cornea, is in agreement with the findings of most authorities. This would make the thickness of the lens 3.6 mm. along the pole. Tscherning, however, has considered this too small an average. He has expressed the opinion that it should be 4.06 mm. Listing gave 4 mm. for the thickness of the lens. The vertex of the posterior surface of the lens does not change its position during accommodation.

In the simplified eye the corneal thickness, its posterior curvature and its index of refraction are all disregarded, and the corneal system is merely the aqueous, separated from air by a single curved surface—the anterior surface of the cornea. Again, in the simplified eye the lens is considered homogeneous throughout, with an index of refraction of 1.413 during relaxation, increasing to 1.424 during accommodation. It is infinitely thin. Its optical center, 5.85 mm. behind the cornea during relaxation, comes forward 0.65 mm. during maximum accommodation.

Gullstrand assumed 7.7 mm. for the radius of curvature of the anterior surface of the cornea, while Helmholtz gave 7.829 mm. and Tscherning, 7.98 mm. Gullstrand's is the nearest of all schematic corneas to the ophthalmometric standard. The radius of curvature of the posterior surface of the cornea is considered to be 6.8 mm. by Gullstrand and 6.22 mm. by Tscherning, the only other investigator who has considered this surface. For his simplified eye, Gullstrand gave 7.8 mm. for the radius of curvature of the cornea.

The radius of curvature of the anterior surface of the lens for both the exact and the simplified eye is 10 mm., which is almost universally accepted for the normal crystalline lens, although Tscherning gave 10.2 mm. for this value. The radius of curvature is 7.911 mm. for the anterior surface of the core lens and 5.76 mm. for the posterior surface. During accommodation both surfaces increase in curvature and become equal, 2.655 mm., making an equal-sided biconvex lens of the thickness of the radius of curvature of a surface. The same is true for the anterior and posterior surface lenses, in which the radii are, respectively, 10 and 6 mm. during relaxation and during maximum accommodation are equal, 5.33 mm.

In the schematic eye the cornea is a convexconcave lens, with front surface power + 48.83 diopters, back surface power — 5.88 diopters and thickness 0.5 mm. in the center, making the equivalent power + 43.05 diopters. This is only 0.03 diopter less than the power of the single curved surface of the corneal system of the simplified eye. These, of course, are the dioptric powers, respectively, of the aphakic schematic and the aphakic simplified eye.

In Tscherning's eye the corneal system, with radii of curvature of 7.98 mm. for the anterior surface and 6.22 mm. for the posterior surface of the cornea, has a dioptric power of 40.98, 2.07 diopters weaker than Gullstrand's corneal system. Helmholtz' schematic corneal system, with a single curved surface, having a radius of curvature of 7.829 mm., has a power of 42.98 diopters, only 0.07 diopter weaker than Gullstrand's corneal system.

In the exact schematic eye the lens system at rest is given a power of 19.11 diopters for air, so as to allow the aphakic eye to be corrected by a lens of between 10 and 11 diopters. The focal length given in the table is the reduced distance for the vitreous and aqueous. The dioptric power of Gullstrand's crystalline lens is lower than the power assumed for the lens by most authorities. The reduced power of Helmholtz' lens for air is about 26.39 diopters (19.75 diopters for vitreous), and that of Tscherning is 21.39 diopters (16.01 diopters for vitreous).

The actual power of the lens is 19.11 diopters; but because of its distance from the cornea it really adds only about 15.6 diopters to the refractive power of the eye. This is the reason too (besides a change in the positions of the principal points) that while the increase of power of the lens during maximum accommodation is 13.95 diopters it adds only 11.93 diopters to the total refractive power of the eye. If the lens were placed 1 mm. nearer the cornea, the shorter separation from the cornea would add 0.65 diopter to the total refracting power of the eye; if 2 mm. nearer, 1.30 diopters, and if 3 mm. nearer, 1.88 diopters. The latter, with crystalline lens almost in contact with the cornea, would

be corrected by less than a — 2 diopter glass in front of the eye. Slight change of position of the crystalline lens, therefore, is hardly a factor in rapid refractive changes.

Gullstrand, by utilizing the core lens in his schematic eye and by a change in the index of refraction during accommodation for the lens in his simplified eye, accounted for the refractive power of the eye sufficient to image an object placed at his chosen near point distances of 102.3 and 100.8 mm., respectively, for his exact and schematic eyes. In the simplified eye the lens in the calculation is infinitely thin, with the vertexes of its two surfaces and its principal points at the optical center. Helmholtz' accommodating schematic eye can account for a change of refracting power of only 6.5 diopters by increasing the radius of curvature of the front surface from 10 to 6 mm. and that of the back surface from 6 to 5.5 mm., without changing his mean index of refraction (1.437). The association of the change in shape of the lens with an increase of the index of refraction during accommodation had never been made with a schematic eye previous to that of Gullstrand. No reasonable comparison, therefore, can be made for his schematic eye with any other.

The removal of the crystalline lens of the schematic eye would require for correction a + 10.74 diopter lens placed so that the posterior principal point of the correcting glass would be 14.5 mm. in front of the cornea. The simplified eye would require a + 10.64 diopter lens placed at the same distance. This does not mean that an axially myopic eye corrected by a — 10.74 diopter lens placed at 14.5 mm. in front of the cornea would be rendered emmetropic after extraction of the lens. On the basis of the constants of the exact schematic eye, aberration being disregarded, this eye is 3.127 mm. longer than the normal. The length of the eye must be taken into consideration in making any calculation. As a matter of fact, a myopic eye corrected by a — 10.7 diopter lens placed 14.5 mm. in front of the cornea would require a + 5 diopter lens placed in the same position to correct it after removal of the crystalline lens.

The same consideration must be given to the hyperopic eye. For example, an aphakic eye which has been corrected with a + 5 diopter lens will have a principal point hyperopia of about 17 diopters and be corrected by a + 13.66 diopter lens placed 14.5 mm. in front of the cornea, and from this, if one allows for the aberration according to Gullstrand, one should deduct about 1 diopter.

Theoretically, an eye corrected by a — 16.7 diopter lens will be emmetropic after removal of the lens. Practically, allowing for aberration, it will have been corrected by a lens of about 18 diopters. A rough estimation of the correction for the aphakic eye can be made

by adding + 10 diopters to one-half the power of the glass necessary to neutralize a previously existing ametropia. For example, an eye which was previously corrected by — 10.75 diopters will require + 10 — 5.37 = + 4.62; one with 5 diopters of hyperopia, + 10 + 2.50 = + 12.50; one with myopia of 18 diopters, + 10 — 9 = + 1, and one with myopia of 24 diopters, + 10 — 12 = — 2 diopters after removal of the crystalline lens.

For the compound system, that is, for the eye as a whole, the dioptric power and the positions of the principal points are the fundamental constants. The anterior nodal point will be found to be 7.078 mm. and the posterior nodal point 7.332 mm. behind the cornea, but Gullstrand considered them of no importance whatever, and they are not included in the table.

It will be seen that the second principal focal point is 0.387 mm. behind the fovea, giving the schematic eye a hyperopia of 1 diopter. Gullstrand has said that he could not make the eye longer than 24 mm., although he thought that, owing to postmortem changes, it may average nearer 25 than 23 mm.

Gullstrand proposed a reduced eye, with an index of refraction of 4/3, a radius of curvature of the cornea of 5.7 mm. and a length of 22.8 mm.; but one with a radius of curvature of 5.5 mm. (22.5 mm. long and principal focal distance 16.5 mm.) is easier to remember and would correspond with Gullstrand's eye sufficiently for rough estimations. According to the accuracy desired in any given case, one or the other of these models can be employed.

Gullstrand stated that the refracting surfaces may be regarded approximately as surfaces of revolution around a common axis. Although in most human eyes the axes of the various surfaces are apparently not strictly coincident, there is no exact proof, and even so, the variations are so slight as to be negligible in effect as far as the position and size of the image are concerned. The eye, therefore, may be regarded as a centered optical system. The axis of this system, or the axis of the eye as it is called, coincides approximately with the straight line joining the vertex of the cornea with a point on the retina between the fovea and the place where the optic nerve enters the eyeball. Therefore, since the visual line is a secondary axis and makes a finite angle with the optical axis, the imagery in the fovea centralis, strictly speaking, belongs to the region of the periphery. However, the resulting astigmatism for an angle of 5 degrees amounts to only about 0.1 diopter.

The principal points are those from which the focal distances are measured. For this reason their positions are important. They are very close, being separated by only about 0.3 mm. The first principal

point, 1.348 mm. behind the anterior surface of the cornea, lies, therefore, in the anterior chamber 0.848 mm. behind the posterior surface of the cornea, roughly about 1.3 mm. behind the anterior surface.

The anterior principal focus lies 15.707 mm. in front of the cornea. This point should be used to determine the size of the retinal image. It is the point where the posterior principal point of the correcting glass should be placed so that it will not affect the size of the retinal image or make a change in the refractive power of the eye. A glass merely shifts the position of the posterior focus of the eye; it does not change the dioptric power.

The position of the fovea determines the refraction of the eye. If it is too far back myopia is present; if too close, hyperopia. Gullstrand has allowed his schematic eye 1 diopter of hyperopia to compensate for the aberration of the dioptric system.

News and Notes

UNIVERSITY NEWS

Postgraduate Work in Ophthalmology and Otolaryngology.—A week of intensive postgraduate work in ophthalmology and otolaryngology will be given in March 1936 at Portland, Ore., under the combined auspices of the Oregon Academy of Ophthalmology and Oto-Laryngology and the University of Oregon Medical School. A course in ocular pathology supervised by Dr. John E. Weeks and Dr. Frank R. Menne, Professor of Pathology of the University of Oregon Medical School, will be offered. Dr. Olof Larsell, Professor of Anatomy, will conduct a course in dissection of the head and neck. Lectures and operative clinics will be presented by Dr. Harry S. Gradle of Chicago, Dr. William L. Benedict of Rochester, Minn., Dr. William P. Wherry of Omaha and Dr. Dean Lierle of Iowa City. Physicians of the northwest who are interested may obtain further information by addressing Dr. A. B. Dykman, Medical Dental Building, Portland, Ore.

GENERAL NEWS

American Board of Ophthalmology.—The American Board of Ophthalmology will hold its 1936 examinations in Kansas City, Mo., on May 11, and in New York in October. Information, syllabuses and applications may be secured from Dr. Thomas D. Allen, assistant secretary, 122 South Michigan Avenue, Chicago. All applications and case reports must be filed sixty days before the date of the examination.

SOCIETY NEWS

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom will be held in London from April 2 to 4, 1936. Mr. J. G. Magor Cardell, Dr. S. H. Browning and an obstetric surgeon will open the discussion on the subject "Ophthalmia Neonatorum."

CORRECTION

In the article by Dr. W. M. Dodge Jr., entitled, "Histopathologic Characteristics of Nutritional Cataract in the White Rat," in the December issue (ARCH. OPHTH. **14**:922, 1935), the statement that the staff of the Nutrition Laboratory of Battle Creek College and Dr. Helen Mitchell directed the feeding of the animals was inadvertently omitted.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Color Sense

SIMULTANEOUS AND SUCCESSIVE COLOR MIXTURES. M. KARBOWSKI, Arch. f. Ophth. **133:** 532 (April) 1935.

Colors can be mixed either by throwing the two different light rays on neighboring cones at the same time or on the same cone in rapid succession. The visual processes in the two cases are, according to Karbowski, fundamentally different. In the second case the eye works under the conditions of "polychromatic monoconoidal" vision (the two different wavelengths stimulating one cone); in the first case each cone receives only one wavelength ("monochromatic monoconoidal" vision).

Experiments of this type were done by Lempicka. She pasted on a rectangular piece of cardboard 2 mm.-wide strips of two differently colored papers, arranging the colors alternately. Seen from a distance of 16 meters the cardboard appeared monochromatic. She also mixed the same two colors with the usual color mixer (rotating disk). The interesting result was that though the proportions of the two colors were the same (i. e., 1:1) in both methods of mixing the resulting mixed color with one method was not the same as with the other. In order to get the same color, the proportions of the two colors on the rotating disk had to be changed to an extent which seemed characteristic for the chosen colors. The rays of greater wavelength seemed to be the stronger ones in the color mixtures, so that, ceteris paribus, less of the light with the longer wavelength was necessary to produce the same mixed color as by the simultaneous method. Similar results were published by Trendelenburg, who compared the results of monocular and binocular simultaneous color mixtures. Tieplov and Yakovleva (*Arch. f. Ophth.* **130:** 463 [Sept.] 1933), however, found that the same quantitative rules applied to each mode of color mixing.

In experiments of his own Karbowski confirmed Lempicka's and Trendelenburg's findings and also showed that if the rectangle carrying the strips of paper was moved farther away from the observer the color of greater wavelength became stronger and modified the hue of the mixture. He explains these differences on the basis of his theory of color vision (*Arch. f. Ophth.* **130:** 469 [Sept.] 1933.

P. C. KRONFELD.

Conjunctiva

CAPILLARY PULSATIONS OF THE CONJUNCTIVA. A. ROLLIN, Bull. Soc. d'opht. de Paris, March 1935, p. 143.

A man of 50 years had an aortic insufficiency with syphilitic aortitis, a spontaneous retinal arterial pulse and pulsations of the capillaries of the conjunctiva. He came for consultation because of the need for glasses in reading. The conjunctival vessels were very hyperemic. The tension of the retinal artery was 110 mm. The intra-ocular tension was 20 but oscillated between 15 and 25 during the pulsations. The visual

acuity was 6/10. The complete examination of the general system, especially of the vascular tree, is outlined. The irregular pulsation of the flowing blood could be seen in the conjunctival capillaries at all times, the movement being of a saccadic nature synchronous with each systole. This was more marked if one observed the vessels at their bifurcations. At the level of the veins this phenomenon entirely disappeared. Rollin advocates the use of the capillaroscope in the examination of all patients with cardiovascular disease.

L. L. MAYER.

THE RÔLE OF ALLERGY IN CERTAIN CONJUNCTIVITIDES. H. LAGRANGE, Bull. Soc. d'opht. de Paris, April 1935, p. 230.

In gaining a concept of the present status of allergy it is essential to revise one's knowledge of general pathology so as to include, owing to the progress of studies of this subject, a humoral state which may or may not be in entire equilibrium. Lagrange states that he does not include in his present study the ocular reaction to tuberculin or the work of Marfan on phlyctenular keratoconjunctivitis. The points in question are anaphylactic sensibility and idiosyncrasy of the conjunctiva. Vernal conjunctivitis has a long history and in the past was frequently confused with an infectious condition. Bacteriologic studies of the gelatinous infiltration of the corneal limbus in the three cases reported by Arlt in 1846, of the hypertrophic perikeratitis of the conjunctiva described by Desmarres in 1847 and of the perilimbic gelatinous infiltrations described by von Graefe in 1854 would have shown no causative organisms. Recent reports of authorities have shown the periodicity of the disease, its irritative phenomena and its relationship to other sensitive states. The various theories are reviewed. Clinical study permits the isolation of a special syndrome, the factors of which may be grouped as follows:

1. Nature of the trouble: The mucous and cutaneous reaction is of a pruriginous character. The sudden appearance and disappearance give a picture of crisis.

2. Environmental states: The disease appears in certain climates, at certain seasons and under certain other environmental conditions.

3. Pathologic associations: Coincident with or frequently alternating with the disease are other anaphylactic manifestations and diverse dystrophic states (endocrine, developmental, pubertal and menopausal).

4. Observations as to infection and habitual histologic reactions in the follicles of the conjunctiva: Bacteriologic researches yield negative results. No inclusion bodies are found in the cells, but eosinophils are present in large numbers.

5. Tests for sensitivity: Cutaneous or intradermal reactions are positive. Prausnitz-Küstner tests give positive proof of passive allergy.

6. Medicamental and therapeutic reactions: The action of epinephrine or ephedrine is immediate. There is an immediate reaction to lens protein. The results of desensitization are positive.

Three experimental facts are necessary to confirm the doctrine: (1) the anaphylactic phenomenon, (2) the protein shock and (3) the colloidoclastic shock. The first was described by Richet and Portier.

The second may be recalled as brought forth by Arthus, and the third was inspired by Widal.

The conclusions necessary to prove that a pathologic state is due to a sensitivity must have in view three aspects: (1) The specific sensitivity analogous to experimental anaphylaxis. (2) The specific sensitivity of allergic type not analogous to experimental anaphylaxis. (3) The nonspecific sensitivity caused by a diathetic instability of the colloidal equilibrium and consisting in another group of allergic reactions.

L. L. MAYER.

Comparative Ophthalmology

ANATOMY AND PHYSIOLOGY OF THE PECTEN IN THE BIRD'S EYE. P. BACSICH and A. GELLÉRT, Arch. f. Ophth. **133**: 448 (Feb.) 1935.

The pecten is a vertical piece of membrane which in the bird's eye projects from the disk into the vitreous. It consists of vessels and supporting interstitial tissue. The latter is not neuroglia nor is it derived from neuroglia. It also contains no nerve fibers. It is most likely of mesodermal origin. The blood vessels have a peculiar structure. The innermost layer is formed by rather high endothelial cells. Then follows a layer of a homogeneous substance which stains blue with Mallory's method. It most likely is hyaline connective tissue. This probably means that the vessels of the pecten have a fixed caliber. This would also explain the absence of nerve fibers, because vasomotor nerves would be useless if the vessel is not capable of motility. The posterior ciliary arteries (in birds) have an unusually thick media and a very solid-looking adventitia consisting of collagenous tissue. Elastic fibers are very scarce. This structure of the posterior ciliary arteries perhaps has the purpose of stabilizing or controlling the blood flow to the pecten.

Bacsich and Gellért believe that the rigidity of the vessels of the pecten precludes the possibility of its taking any part in the production of fluid for the eye. They are inclined to join others who consider the pecten a heating device for the interior of the eye. The high altitudes which are reached by many birds probably entail excessive losses of heat for all exposed organs like the anterior segment of the eye. Thus the presence of a special heating plant in the posterior half of the eye where homoiothermic conditions are essential for proper functioning of the retina seems quite plausible.

P. C. KRONFELD.

THE INVOLUTION OF THE TUNICA VASCULOSA LENTIS IN MONKEYS. M. BÜCKLERS, Arch. f. Ophth. **134**: 48 (May) 1935.

Bücklers studied the involution of the fetal vascular system of the lens in fifteen Javanese macaci (Macacus irus mordax). At birth the pupillary membrane consists of a corona of vessels on the iris which sends a few branches toward the pupillary border. In premature monkeys these branches still reach the capsule of the lens. The ramification of the hyaloid artery on the posterior surface of the lens is always clearly visible at birth. The communication between the anterior and the posterior vascular system, the membrana capsulopupillaris, presents itself in the form of radial hairpin-like vessel loops on the peripheral

portion of the anterior capsule of the lens. Four weeks after birth the fetal vascular system has been completely absorbed. The embryonic lens sutures are of the same shape and direction as in man.

P. C. KRONFELD.

Congenital Anomalies

CONGENITAL BILATERAL MELANOSIS OCCURRING IN THE EYE AND FACE: A CONTRIBUTION TO THE PROBLEM OF CONSANGUINITY. P. KIEWE and I. MANEFF, Ann. d'ocul. **172**: 723 (Sept.) 1935.

Ocular hyperpigmentation has interested ophthalmologists at all times, and their interest has increased with the study of hereditary conditions of the eye. The number of cases in which pigmentation has been proved to have a hereditary origin are few. Franceschetti (according to Schieck and Brückner) noted only three publications on cases of hereditary pigmentation of the eye: those of Bourquin, Fleischer and Selter.

The case reported is that of an adult man, 64 years of age, who had marked pigmented areas over the face, especially about the eyes. The lids in particular were pigmented, especially along the edges, where there were fine brownish-black spots that tended to come together. The palpebral conjunctiva presented a similar picture, and the deeper parts were more involved than the superficial. Examination with the slit lamp showed the pigmentation to be in the subepithelial layer. The cornea presented only arcus senilis. General examination revealed nothing of interest except a pigmentation on the trunk and extremities similar to that of the face. A son of this patient, a boy of 8 years, presented a similar condition.

The article is illustrated with a good photograph of the patient, colored drawings of the lesions in the skin and a chart illustrating the consanguineous relationship in such cases.

S. H. MCKEE.

RARE MALFORMATIONS OF THE IRIS. H. RIEGER, Arch. f. Ophth. **133**: 602 (April) 1935.

Two cases are reported in great detail: A girl 10 years of age showed an opaque zone in the cornea next to the limbus and persistent mesodermal tissue in the filtration angle, on the posterior surface of the cornea and on the anterior surface of the iris. This tissue had led to strand-shaped adhesions between the cornea and the iris. The mesodermal portion of the iris was underdeveloped and atrophic because of glaucoma. One pupil was pear-shaped and the other slit-shaped.

A girl 25 years of age had pear-shaped pupils and also strands of tissue which originated from the angle of the anterior chamber and formed an adhesion between the cornea and the iris, ending up at the most derounded (most eccentric) portion of the pupillary border. This patient had no glaucoma.

Rieger believes that all cases of congenital hypoplasia of the stroma of the iris, irrespective of the shape and position of the pupil, belong to one group in which the same abnormal development has taken place, namely, a persistence of the fine strands of protoplasm which connect the fetal pupillary membrane with the cornea and the anterior projection of the uvea. The cases of embryotoxon posterius corneae (Axenfeld),

by which term various types of congenital opacities of the deep peripheral portions of the cornea are meant, belong to the same group. The name "dysgenesis mesodermalis corneae and iris" is recommended for this group.

P. C. KRONFELD.

Cornea and Sclera

FISTULA AT LIMBUS (SCLEROMALACIA PERFORANS). C. T. EBER, Am. J. Ophth. **17:** 921 (Oct.) 1934.

Eber reports the case of a man, aged 65, whose left eye showed a spastic entropion of the lower lid and at the upper nasal limbus of the cornea a white cystlike area, from 4 to 5 mm. wide, extending 6 mm. along the limbus and about 2 mm. over it, encroaching on the cornea. At operation it was noted that the sclera beneath this bleb remained moist, and on close observation a small opening was seen just behind the limbus at 10 o'clock, from which exuded a clear fluid (evidently aqueous humor). Van der Hoeve reported four cases of what he termed "a typical disease of the sclera," which exhibits various-sized holes in the sclera without dominant signs of inflammation and is often accompanied by rheumatic polyarthritis. He called it scleromalacia perforans.

W. S. REESE.

KERATOPLASTY. R. CASTROVIEJO, Am. J. Ophth. **17:** 932 (Oct.) 1934.

Castroviejo reports seven cases in which corneal transplants were used. He shows photographs of the corneas before and after operation, and gives the following summary and conclusions:

"It has been the purpose of this paper to point out that homokeratoplasty in human beings can be successfully accomplished when a suitable technic is followed.

"The principal features of the technic already given in detail are: (1) to obtain a rectangular graft, 4 mm. square, corresponding exactly in size and beveled edges in both the transplant and the opening in the cornea of the recipient, with the anterior surface of the graft larger than the posterior surface; (2) the use of conjunctival flaps to hold the transplant in position, giving a gentle uniform pressure, accelerating the process of healing, and furnishing necessary nutrition while cicatrization takes place during the first few days. The conjunctival flaps also make a water-tight union favoring the prompt restoration of the anterior chamber, and finally, they offer the best possible protection for the graft and the eye in case the former becomes partially detached or does not heal.

"The technic of partial penetrating keratoplasty, such as the one described above, will give the best results in those eyes in which the whole pathology is limited to the cornea, the leucoma being not very dense, and in which there are zones of little- or nonscarred cornea surrounding the transplant. In eyes with dense corneal leucomas in which the transplant is entirely surrounded by dense scar tissue, in those with aphakia and loss of vitreous during the operation, hypertension, etc., the results cannot be expected to be so brilliant, but inasmuch as the vision in these eyes is limited to light perception and projection, or only

perception, and the corneal transplantation offers the only means to improve the vision, operation is advisable.

"Eyes with heavy scarring of the conjunctiva around the cornea are predisposed to glaucomatous attacks, hence it would be good judgment in these cases to perform a large iridectomy previous to corneal transplantation.

"Transplants should be obtained from enucleated eyes in which the cornea has been left entirely unaffected. Transplants should not be taken from glaucomatous eyes because the cornea is always more or less affected by the pathological process and the transplant has a tendency to remain edematous and afterwards, nebulous.

"Enucleation of the donor's eyes should be performed, if possible under general anesthesia to avoid injury of the corneal epithelium which almost always occurs when local anesthesia is used. Eyes of young people seem to furnish better material for transplantation than those of older persons.

"The importance that should be ascribed to blood grouping in corneal transplantation is to be determined when the operation has been performed in a large number of cases. Blood grouping was made in the six last cases reported and in each instance it was found to be different in the donor from that in the recipient.

"In examining patients with dense leucomas extending over the whole area of the cornea, posterior and anterior transillumination is the most valuable method."

W. S. REESE.

TREATMENT OF RODENT ULCER OF THE CORNEA. A. BRÜCKNER, Bull. Soc. d'opht. de Paris, April 1935, p. 255.

A man of 36 years had been treated for half a year without success. The organisms were Bacillus subtilis and Bacillus xerosis. Iodine was of no avail. A fistula-making iridosclerectomy of the Lagrange type caused disappearance of the pain and arrest of the ulcer. With reappearance of the ulcer after a month's time a radical excision of its advancing borders with a grattage including the deeper vessels caused permanent healing.

L. L. MAYER.

IONIZATION WITH POTASSIUM IODIDE AND SODIUM SALICYLATE AND ITS CLEARING EFFECTS ON CORNEAL OPACITIES. JUAN MANUEL VILA ORTIZ JR., Arch. de oftal. hispano-am. **35:** 25 (Jan.) 1935.

After a more or less detailed account of the theory and practice of ionotherapy, a report is made of twenty-nine cases in which this method of treatment was employed in connection with corneal opacities. With potassium iodide in concentrations of from 1:200 to 1:400, a current of from 3 to 5 milliamperes and a duration of from ten to twenty minutes good results were obtained in 50 per cent of the cases. With sodium salicylate in a concentration of 1:200, a current of from 2 to 5 milliamperes and a duration of from fifteen to twenty minutes, two to three times a week, success was obtained in 30 per cent of the cases. With both solutions combined success was obtained in 100 per cent of the cases. These opacities, however, were superficial and recent.

C. E. FINLAY.

Experimental Pathology

STUDIES ON VASCULARIZATION OF THE CORNEA: HISTOLOGIC CHANGES ACCOMPANYING CORNEAL SENSITIVENESS. L. A. JULIANELLE and H. D. LAMB, Am. J. Ophth. **17**: 916 (Oct.) 1934.

When certain bacteria are instilled repeatedly into the conjunctival culdesac of the monkey or the rabbit, following gentle scarification of the cornea, a hypersensitive reaction is stimulated which is characterized by corneal cloudiness and vascularization. Changes of the same type follow repeated intracorneal injections of sterile crystalline egg albumin or bacterial nucleoproteins; hence such changes are not due primarily to infection. Following experiments, Julianelle and Lamb conclude: "While it is obvious that no evidence has been adduced to show that the corneal reaction of hypersensitiveness is related in genesis to the corneal reaction of trachomatous pannus in man, nevertheless both conditions exhibit certain characteristics in common. Therefore the comparisons and contrasts between the two reactions are described, to point out the essential analogies until further studies are undertaken to reveal a possible genetic relationship." W. S. REESE.

NEW EXPERIMENTAL DATA CONCERNING THE PATHOGENESIS OF CATARACT. N. K. MONYUKOVA and M. J. FRADKIN, Arch. f. Ophth. **133**: 328 (Jan.) 1935.

A certain amount of cevitamic acid, or vitamin C, has been found in the aqueous of the normal eye. If the lens becomes cataractous the amount of cevitamic acid in the aqueous decreases. The progression of the cataract seems to parallel the decrease in the cevitamic acid. The authors of this paper have tried to determine whether that change in the composition of the aqueous precedes or follows the development of the cataract.

Guinea-pigs were fed a scurvy-producing diet. When the scurvy was well advanced some animals presented partial cataracts of a certain morphologic type. In others in which the lens had remained clear the aqueous was withdrawn once without injuring the lens. In these animals cataracts developed within twenty-four hours after the paracentesis. The second aqueous has been known to contain very little, if any, cevitamic acid. The authors therefore interpret the results of their experiments as follows: If the guinea-pig is deprived of the necessary vitamin C in its food it often acquires a cataract. This cataract can be produced very acutely if in the guinea-pig which suffers from scurvy the cevitamic acid content of the aqueous is lowered still further for a short period (by puncture of the anterior chamber). Therefore the authors believe that the decrease in the cevitamic acid content of the aqueous in cataractous eyes is at least one of the causes and not a result of the cataract.

They explain the fact that the aqueous contains more cevitamic acid than the blood by assuming a selective permeability of the blood-aqueous barrier for this substance. If this mechanism by means of which a

high amount of cevitamic acid is maintained in the eye is disturbed cataract should develop. This is perhaps the mechanism underlying senile cataract.

P. C. KRONFELD.

FURTHER EXPERIMENTAL DATA CONCERNING THE PATHOGENESIS OF CATARACT. N. K. MONYUKOVA and M. J. FRADKIN, Arch. f. Ophth. **133:378** (Feb.) 1935.

The authors base some very comprehensive conclusions on the fact that several female guinea-pigs which received a scurvy-producing diet during the last three weeks of pregnancy gave birth to a number of animals that exhibited congenital opacities of the lens.

P. C. KRONFELD.

EXPERIMENTAL INTERSTITIAL KERATITIS. Y. YOKOTA, Arch. f. Ophth. **133:** 382 (Feb.) 1935.

Yokota has been mainly interested in finding out by what route spirochetes inoculated into the anterior chamber of the rabbit invade the cornea. The clinical picture of the interstitial keratitis which follows the inoculation varies. In some animals the keratitis starts out from the limbus and then progresses across the cornea. In others a localized tumor-like granuloma develops near the limbus. In these cases the inflammatory symptoms are pronounced. In the third group of animals the experimental keratitis takes the course of a deep disk-shaped infiltration in the center of the cornea. In each of these groups the limbus seems to be the place where the spirochetes enter the cornea. After inoculation into the anterior chamber the spirochetes are apparently carried toward the angle of the chamber where they first invade the tissue. In the lymphatics of the limbus they seem to find favorable conditions for multiplication. From there they invade the cornea. Thus it is possible that in early stages of experimental keratitis the spirochetes are only found in the superficial layers of the cornea.

P. C. KRONFELD.

General

OCULAR DISORDERS ASSOCIATED WITH THE WISDOM TOOTH. C. B. HENRY, Brit. J. Ophth. **19:** 378 (July) 1935.

Henry cites from the literature and from his own experience cases to show that a perfectly healthy but unerupted wisdom tooth may produce great disturbances in the eye, which must be attributed either to direct pressure on neighboring nerves or to some reflex irritation. MacCallan supplies the notes on a case of diplopia and paresis of the right inferior rectus muscle. Three days after the extraction of unerupted right upper and right lower third molars, conditions became normal.

Clark adds the history of a young woman, aged 20, who had constant ocular pain for four years, followed by migraine. Esophoria developed, and the eye was operated on without relief. Roentgenograms showed a right lower wisdom tooth emerging from the alveolus and directed a little forward. The tooth in front of it was removed to make room and so relieve the pressure, and there was an immediate improvement.

Roller reports a case of bilateral iritis in a woman aged 35 years. Roentgenograms revealed healthy teeth except for an unerupted left lower wisdom tooth. After its extraction improvement set in, and a cure followed. The roots of the tooth were very close to, if not actually touching, the inferior dental nerve, which seems to show that the condition of the eye may have been due to the reflex irritation of pressure on the nerve.

The article is illustrated.

W. ZENTMAYER.

EXPERIENCE WITH ACLASTIC GLASSES. A. TERSON, Bull. Soc. d'opht. de Paris, January 1935, p. 26.

Terson describes a biconcave protective lens made from a new cellulose compound. It is noninflammable and nonshatterable. It has been accepted by the French Association for the Prevention of Blindness.

L. L. MAYER.

FUNGI GROWN FROM THE CONJUNCTIVA, CORNEA AND LACRIMAL PASSAGES. A. FAZAKAS, Arch. f. Ophth. **133:** 461 (Feb.) 1935.

A study of fungi in 1,017 cases of various external diseases of the eye was undertaken. The mediums used were maltose-agar and dextrose-peptone. Various types of fungi were found. Fungi were grown from 35.8 per cent of the diseased eyes and from 24.3 per cent of normal controls. "Positive fungus cultures are therefore much more frequent in diseased eyes. The presence of the fungus may greatly influence the course of the ocular disease."

P. C. KRONFELD.

General Diseases

METHYLIC ANTIGEN AND TUBERCULOSIS OF THE EXTERNAL SEGMENT. L. Genet, Bull. Soc. d'opht. de Paris, January 1935, p. 44.

The diagnosis of ocular tuberculosis in cases of inflammatory lesions involving the sclera and cornea is always difficult. This disease is without the characteristic features of all of the other inflammatory manifestations of tuberculosis, such as nodules and caseation. One depends for the most part on eliminating other etiologic factors and finally on the therapeutic test.

Genet cites in detail a case in which the usual characteristics of tuberculous disease involving the cornea and sclera were observed and demonstrates the excellent results obtained by the use of the methylic antigen.

L. L. MAYER.

Instruments

A KNIFE FOR CORNEAL GRAFTING. H. B. STALLARD, Brit. J. Ophth. **19:** 459 (Aug.) 1935.

Stallard has devised a knife for shelving the donor's graft and the corneal bed of the recipient to correspond. The details of his procedure and the use of the knife are given.

The article is illustrated.

W. ZENTMAYER.

Lacrimal Apparatus

CONCRETIONS IN A LACRIMAL CANALICULUS CAUSED BY ACTINOMYCES. A. CHARLOTT RUYS, Brit. J. Ophth. **19:** 385 (July) 1935.

The case was observed and the condition diagnosed by Hagedoorn. The patient was a country woman aged 30 years. There had been epiphora and a discharge for two years. A little swelling and redness were noticed at the site of the right lower canaliculus. Smears of the pus showed a mass of fine filaments and many leukocytes. A study of the filaments disclosed that they were of a strain belonging to the genus Actinomyces.

After the diagnosis the canaliculus was slit. The duct contained several clumps of soft yellowish material. After their removal there was no further complaint. The clumps consisted of the same fungus.

The article is illustrated. W. ZENTMAYER.

Lens

RAPIDLY DEVELOPING CATARACTS AFTER DINITROPHENOL. W. W. BOARDMAN, J. A. M. A. **105:** 108 (July 13) 1935.

Boardman reports two cases in which cataracts developed rapidly in patients who were taking dinitrophenol for obesity. The first patient was a woman aged 50 years. She had been taking dinitrophenol off and on for about eighteen months to within a week of the time that incipient cataract was diagnosed.

The second case occurred in a woman aged 36. After the interrupted use of dinitrophenol over a period of about sixteen months cataract developed rapidly, and within one month's time vision in the right eye equaled perception of light.

Four other cases occurring in the practices of other physicians are mentioned.

Boardman believes that, as the occurrence of cataract at this age is extremely rare and the only common factor in these cases has been the use of dinitrophenol, the administration of the drug is definitely contraindicated until the question of its relationship to the rapidly developing cataracts in young women is settled. W. ZENTMAYER.

CATARACTS FOLLOWING THE USE OF DINITROPHENOL. W. D. HORNER, R. B. JONES and W. W. BOARDMAN, J. A. M. A. **105:** 109 (July 13) 1935.

The authors give the notes on three cases in which rapidly developing cataract occurred during the administration of dinitrophenol for obesity. The patients were all women. In three other patients using the same drug the lenticular changes could not be attributed to a specific action.

W. ZENTMAYER.

CATARACT DUE TO EXPERIMENTAL ASPHYXIA. G. BIOZZI, Arch. f. Ophth. **133:** 423 (Feb.) 1935.

The physiologist G. Amantea (University of Rome), in the course of his extensive studies on reversible asphyxia, noticed first the develop-

ment of bilateral cataract in a rat which had been incompletely asphyxiated every day for a month. The technic was as follows: The animal (weighing from 150 to 250 Gm.) was put in an air-tight glass container of 3 liter volume. By careful observation of certain reflexes one was able to determine how long the animal could stand this asphyxia. This period was fairly constant in white rats.

Biozzi has studied the various ocular changes which occur during experimental asphyxia of a degree which enables the animals to recover in the large majority of cases if brought into the air again. These changes consist of dilatation and increased permeability of the uveal vessels. The protein content of the aqueous increases. A formed exudate may be found in the anterior chamber. A cataract develops in the superficial layers of the cortex. All these changes are reversible even if the asphyxia is repeated many times. He considers the cataract a result of the uncompensated acidosis that prevails during asphyxia.

P. C. KRONFELD.

Methods of Examinations

ARTIFICIAL DAYLIGHT FOR TESTING THE COLOR SENSE WITH ISHIHARA'S CHARTS. P. KISSIN, Arch. f. Ophth. **134:** 38 (May) 1935.

Ishihara's pseudo-isochromatic charts have been designed for "good daylight." If they are used in ordinary electric light the isochromatic effect of some of the charts is lost and the test becomes less sensitive. L. Wentmann, apparently a physicist, employed by the Soviet Government, has devised a rather complicated liquid filter which the person whose color sense is to be tested in electric light wears in a trial frame. The filter contains various blue, bluish-green and violet dyes. The results obtained with this filter have been rather encouraging, as the responses of color-blind persons tested in daylight and then in electric light plus the filter were essentially the same. Kissin hopes to get still better results with other filters.

P. C. KRONFELD.

Ocular Muscles

SOME REMARKS ON THE RECESSION OPERATION FOR SQUINT. J. G. MILNER, Brit. J. Ophth. **19:** 448 (Aug.) 1935.

The object of the paper is to show the value of the operation of recession of the internal rectus muscle for convergent concomitant squint. The results in the fifty cases are compared with the results in a number of cases in which surgical intervention was by other methods and by several other surgeons.

In the latter group of cases, in the combined operation of advancement with tenotomy, the eyes were straight (within 5 degrees) in only 54.7 per cent of the cases. Tenotomy for divergent squint appears to be very satisfactory. Advancement has little hope of success. Of the series of fifty cases in which the eyes were operated on by recession, in thirty-seven, or 74 per cent, the eyes were straight (within 5 degrees) after periods varying from three months to three years; in ten cases the eyes were straight only with glasses and convergent about 10 degrees

without glasses. The average angle of reduction was 24 degrees. Of the thirteen cases in which the eyes were not straight, in nine the squint was alternating, and of these nine, in seven the original angle of squint was 30 degrees or more.

The author describes the method employed in operating.

W. ZENTMAYER.

THE RESPONSE OF CROSS-EYED PATIENTS TO VESTIBULAR EXAMINATION: NORMALLY INCITED NYSTAGMUS; HYPO-EXCITABILITY OF THE VESTIBULE; ASYMMETRICALLY INCITED NYSTAGMUS. G. JAYLE, Bull. Soc. d'opht. de Paris, February 1935, p. 90.

The response of cross-eyed patients to vestibular examination is relatively little known. Jayle in conjunction with Aubaret and Appaix gave their first report before the Ophthalmological Society of the Southeast. The approved rotatory and caloric tests were made. The results obtained have been interpreted according to the outline given by Baldenweck. Thirty-one cases were studied. They may be divided into three large categories:

1. Normal vestibular response—thirteen cases
   (*a*) Four cases of alternating convergent strabismus
   (*b*) Three cases of convergent strabismus with unilateral amblyopia
   (*c*) Five cases of divergent strabismus with unilateral amblyopia
   (*d*) One case of alternating divergent strabismus
2. Hypo-excitability of the vestibule—eighteen cases
   (*a*) Four cases of alternating convergent strabismus
   (*b*) Five cases of convergent strabismus with unilateral amblyopia
   (*c*) Three cases of divergent strabismus with unilateral amblyopia
   (*d*) Six cases of alternating divergent strabismus
3. Asymmetrically invoked nystagmus—none
   (Cases have been reported in which the nature of the nystagmus changes.)

Jayle believes that hypo-excitability of the vestibule may indicate previous paralysis of the musculature.

L. L. MAYER.

Physiologic Optics

COMPUTATION OF THE AXIAL LENGTH OF THE EYE. L. PAUL, Arch. f. Ophth. **133**: 254 (Jan.) 1935.

This paper is a continuation of previous papers by Paul on localizing ophthalmoscopy. The axial length of the eye can be computed if all the optical elements are known. Of these, the refractive power of the lens and the depth of the anterior chamber are difficult to measure. Paul has tried to determine how accurately the axial length can be computed from the refractive power of the cornea and the total refractive error alone, without considering the variations in the refractive power of the lens. The axial length thus computed he calls the apparent one in contradistinction to the true axial length. In the aphakic eye the true axial length can be calculated. He determined the apparent

axial length in thirty-three eyes, later on removed the cataract and then computed the true axial length. In twenty-one eyes (64 per cent) the difference between the true and the apparent axial length was less than 2 per cent. If the highly myopic eyes were eliminated, a discrepancy between the two axial lengths of less than 2 per cent was found in 75 per cent of the eyes. Therefore he believes that in the large majority of cases the apparent axial length differs from the true one only by from 1 to 2 per cent. He gives tables and graphs which facilitate the computation of the apparent axial length. P. C. KRONFELD.

Physiology

A STUDY OF THE CENTRAL AND PERIPHERAL LIGHT AND DARK ADAPTATIONS WITH VARYING BACKGROUNDS. R. PICKARD, Brit. J. Ophth. **19:** 481 (Sept.) 1935.

Pickard says that if the light threshold is defined as the least amount of light seen on a black background, and the light difference as the least amount of black seen on a white background, it is obvious that these extremes are joined together by an infinite number of intermediate positions from each of which a departure may be made in either direction with sensations lighter or darker than the background.

In the present paper the least differences in either direction at intermediate points, as well as in the one possible direction at the two extreme positions, are investigated. The inquiry is directed to the peripheral as well as the central capacity for discrimination and to the time required for adaptation. The paper, which is of considerable length, gives in detail the methods pursued and the results obtained. There is much that is technical and does not lend itself to abstracting.

W. ZENTMAYER.

EXPERIMENTAL STUDIES OF THE RELATION BETWEEN THE PRESSURE IN THE RETINAL VEINS AND THE INTRACRANIAL PRESSURE. J. G. LINDBERG, Arch. f. Ophth. **133:** 191 (Jan.) 1935.

In these experiments the pressure in the retinal veins was determined with a new type of dynamometer which might be called a suction speculum. It has the shape of a drum with a brim at its open base shaped to fit over the anterior segment of the rabbit's eye, and a glass window in front. This drum is connected through a manometer with a suction device. The observer regulates the vacuum by holding the free end of the system in his mouth and closing or opening it with his tongue (like Nugent's modification of Barraquer's suction device). The fundus is seen by indirect ophthalmoscopy through the glass window of the speculum. The greater the vacuum in the drum, the more it presses on the globe and increases the intra-ocular pressure. The venous pressure is determined by producing a vacuum in the drum just sufficient to make the retinal veins collapse. At that moment the pressure is read on the manometer. These readings are relative ones, but can be transposed into absolute ones by applying the suction speculum and a Schiötz tonometer to the same eye at the same time (one over the cornea, the other over the sclera). The intracranial pressure is varied by inflation of air into the cranial cavity. For this purpose he has worked out a very neat technic.

His results are: The venous pressure in the retina parallels the artificially raised intracranial pressure very closely. These experiments confirm Baurmann's view that the venous pressure in the retina usually equals the intracranial pressure. At an intracranial pressure of 40 mm. of mercury the difference in pressure between the retinal arteries and the veins reaches a minimum. At this pressure the phenomena of stagnation or choking are most likely to occur. If the intracranial pressure is raised above 40 mm. of mercury the blood pressure rises automatically to prevent anemia of the brain. Lindberg has not been able to observe in his rabbits any fundus changes which could have been interpreted as beginning choked disk. Even a pressure of 40 kept up for three hours or a pressure of 60 maintained for short periods does not change the ophthalmoscopic appearance of the disk.

P. C. KRONFELD.

Refraction and Accommodation

POSTTRAUMATIC MYOPIA AND APHAKIA. F. TERRIEN and P. DUMONT, Bull. Soc. d'opht. de Paris, March 1935, p. 140.

A girl of 19 years wished to have her eyes examined to determine whether she could go without her glasses. She was wearing a +3.0 diopter sphere for the right eye and a —12.0 diopter sphere for the left. The history revealed that the girl was myopic in both eyes at birth. A picture taken at the age of 9 years shows her with both eyes corrected by myopic lenses. There was no history of myopia in her antecedents. At the age of 9 she was hit in the right eye with great violence by her cousin with a rock. She remembers only that the eye was irritable for about one year. An ophthalmologist prescribed a wash. In 1934 she was given her present correction. She explained that she viewed distant objects with her right eye and read near print with the left eye. She had never had diplopia. Examination revealed that the lens of the right eye was missing, but there was no evidence of an operation. A capsular opacity persisting in the form of a cross was seen in the inferior nasal quadrant. Some fine lenslike material was noted in the superior and temporal regions. With the diploscope there was no binocular vision but an alternating monocular vision. Simultaneous vision was in evidence with the Maddox rod. The fundi were normal. A subjective test brought forth that the image for the right eye was one-fifth larger than that for the left. The authors offer this case as a plea for extraction of the lens in young persons with high myopia and normal fundi.

L. L. MAYER.

NEW WAYS IN THE STUDY OF ACCOMMODATION: V. THE VITREOUS IN THE ACCOMMODATING EYE. A. VON PFLUGK, Arch. f. Ophth. **133:** 545 (April) 1935.

Von Pflugk is known for numerous papers dealing with the mechanism of accommodation. This time he studied the mechanical processes which take place in the anterior vitreous during accommodation. He used fresh eyes from the abattoir and made the vitreous directly visible from the outside by cutting windows into all three tunics of the eye in the equatorial region. He then injected one drop of a dye into the

anterior vitreous, midway between the ciliary body and the posterior pole of the lens. If he then pressed against the ciliary body from the outside in radial direction the dye droplets moved toward the lens capsule. Pressure exerted at any other place (anterior or posterior pole of the eye) did not move the droplet. This displacement of the anterior vitreous on pressure in the direction from the ciliary body, observed on freshly enucleated eyes, supports his view that the accommodating lens changes its shape under the influence of pressure on the part of the vitreous. This pressure acts chiefly on equatorial, i. e., young and elastic portions of the lens. To accomplish this change in shape the lens substance must be soft and plastic, and the vitreous must possess a certain amount of solidity. If the vitreous becomes liquefied its modeling action on the lens is bound to decrease. Von Pflugk assumes that presbyopia is caused not only by increasing rigidity of the lens but also by decreasing solidity of the vitreous. P. C. KRONFELD.

Retina and Optic Nerve

OPTIC NEURITIS IN THE COURSE OF TREATMENT BY INJECTIONS OF PENTAVALENT ARSENICALS. P. VEIL, Bull. Soc. d'opht. de Paris, February 1935, p. 71.

Two cases are reported by Veil. One patient was tabetic and was treated with acetylarsan; the other, in whom Ambard's constant was greatly elevated, was treated with arsaminol. The first patient was a woman of 50 years with normal pupils, fundi, visual fields and visual acuity in 1929. In 1933 after the third injection of acetylarsan in seven hours' time there occurred a mistiness of both eyes. The right eye showed an immobile dilated pupil; there was perception of light with poor projection. The optic nerve head was slightly discolored. The arteries maintained their normal caliber. The left pupil was not dilated but reacted slowly to light. Vision was only 0.4. The field of vision was reduced to within 6 degrees of the fixation point, but there was no central scotoma. The papilla was slightly discolored, but the arteries were normal. The patient was deaf. Treatment was discontinued and replaced by injections of acetylcholine and sodium hyposulfite. After two years the right eye has no perception of light and the nerve head is white. The left eye has an acuity of 0.4 and visual fields in the neighborhood of 10 degrees. The deafness has disappeared.

The other patient, a 43 year old woman, had her reaction after the tenth injection. The right eye remained normal. The left eye had a visual acuity of 0.2; the fundus was normal but the field was contracted without central scotoma. Treatment with acetylcholine brought the vision back to normal; the fields became normal, but the papilla became discolored. Pupillary reactions to light, which had been entirely absent after the attack, returned to normal after one year's time.

L. L. MAYER.

SOME CASES OF HYPOTENSION OF THE RETINAL ARTERY. M. LAIGNIER, Bull. Soc. d'opht. de Paris, March 1935, p. 151.

Bailliart and Tillé were able to demonstrate that a retinal arterial hypotension is of as much importance as the hypertension. Laignier

reports concerning a patient who complained of difficulty in reading printed characters. The entire ocular examination revealed no abnormalities except that, for the left eye, a retinal arterial tension of 28 mm. was recorded, while the right was normal at 55 mm. In such a case one must think of a spasm of the left retinal artery. General physical examination revealed an alexia with a left homonymous hemianopia. The visual acuity was 10/10 for either eye, and the pupillary light reflex was preserved for the hemianopic area. Reduplication of the first heart sound at the apex, a dilated and hypertrophied left chamber of the heart and a relative aplasia of the right carotid artery were the physical symptoms.

A second patient noticed spots before the right eye and loss of memory. The visual acuity was 10/10 for either eye. The retinal arterial tension of the right eye was 30 mm.; that of the left, 60 mm. There was a systolic murmur at the base with an aortic murmur and a diastolic bruit. The mitral valves were insufficient. Graphs of the carotid pulse are shown. The importance of the lowered tension of the retinal arteries and an explanation of the cerebral lesions are emphasized.

L. L. MAYER.

LESIONS OF THE MACULA OF CAPILLARY ORIGIN. BAILLIART, SCHIFF-WERTHEIMER and MIROUX, Bull. Soc. d'opht. de Paris, April 1935, p. 180.

A woman 64 years old complained of dimness of vision of both eyes. The difficulty had been present for two months; it began suddenly with phosphenes (light flashes of very rapid occurrence), and later black patches were seen. The visual acuity of each eye was reduced to 3/10. The fundi were normal except that in both macular areas there was a white exudate, refractile and raised about 1 diopter. Near the margins, which were more or less regular, a destructive process in the retina had made the underlying intact choroid visible. In the destroyed area were numerous arterioles dilated and transformed into small white cords. On compression of the globe eight or nine apparently thrombosed arterioles were seen. There were a few discrete opacities on the posterior surface of each lens. Aside from a very slight rise in general arterial blood pressure all examinations gave normal results. The authors draw attention to the preservation of occupational visual acuity. A photograph is shown.

L. L. MAYER.

INFLUENCE OF AN EXCISION OF THE SCLERA AFTER LINDNER ON THE ANTERIOR SEGMENT OF THE EYE. F. RAMACH, Arch. f. Ophth. **133**: 321 (Jan.) 1935.

For certain cases of retinal detachment with very unfavorable prognosis, Lindner (*Ztschr. f. Augenh.* **81**: 277, 1933) advocated excision of a ring-shaped strip of sclera, from 2 to 6 mm. wide, from the region of the equator. This was usually done in two sittings. Thus the sclera was shortened more or less evenly in all meridians. Lindner performed this operation on twelve eyes. These operations produced some rather unexpected effects on the shape and function of the anterior half of the eye. The palpebral fissure became narrower, and the apex

of the cornea receded somewhat. The curvature of the cornea was always altered by the operation. The anterior chamber became shallower. The sensitivity of the cornea was temporarily impaired.

P. C. KRONFELD.

PATHOLOGIC OBSERVATIONS AFTER IGNIPUNCTURE (GONIN). E. F. LEWKOJEWA, Arch. f. Ophth. **133**: 467 (Feb.) 1935.

A woman 40 years of age had been operated on by Lindner for a retinal detachment in a highly myopic eye. Three operations were performed within two months. Fifteen months later the atrophic eye was enucleated because of pain. One ignipuncture had been made 9 mm. from the limbus. There the tract of the cautery was still visible. It ended in the pars plana of the ciliary body. From there connective tissue membranes connected it with the equator of the lens. The sclera was still in a state of necrobiosis. This area extended forward to the scleral spur. There was a diffuse infiltrative uveitis. The retina was detached except in two places which corresponded to areas of operation. The retina showed multiple fixed folds, extensive degeneration and the results of massive hemorrhages.

From the anatomic findings Lewkojewa concludes that the operations were too severe. The cautery attracted the retina with such force that a flat reattachment became impossible. Much too large a portion of the retina became bound to the area of operation. This led to further pulling on the remaining retina and to secondary postoperative tears. One operation was made too far forward; thus the ciliary body and the lens were injured.

P. C. KRONFELD.

PATHOLOGIC OBSERVATIONS AFTER IGNIPUNCTURE (GONIN): REMARKS IN ANSWER TO THE PAPER BY E. F. LEWKOJEWA. K. LINDNER, Arch. f. Ophth. **133**: 476 (Feb.) 1935.

Lindner stresses the point that the eye under discussion was the only one of his 1929 series that became atrophic. Cauterizations which were done just as near the limbus and just as long as in the case under discussion led to complete and permanent reattachment in many cases. The cautery was always directed slightly backward while going through the sclera. The topographic relations described by Lewkojewa do not represent the status after the last operation but rather the result of excessive shrinking of membranes which took place later on. The main reason why operations for retinal detachment fail is that severe intraocular hemorrhages occur shortly or some time after the operation. But even then permanent reattachment is not impossible if the retina has returned to its normal position immediately after the operation. One should not forget that some eyes with retinal detachment become atrophic without any surgical intervention.

P. C. KRONFELD.

THE CIRCULATION OF THE BLOOD IN THE RETINA IN A CASE OF RETINITIS EXUDATIVE EXTERNA. H. SERR, Arch. f. Ophth. **133**: 508 (Feb.) 1935.

Serr describes a typical case of retinitis exudativa externa in a youth 18 years of age. The retina lay so far forward in the vitreous that it

could be easily examined with the slit lamp. There were numerous miliary aneurysms and arteriovenous anastomoses. The circulation in these anastomoses is described in detail. P. C. KRONFELD.

THE MECHANICS OF RETINAL DETACHMENT IN CASES OF INTRA-OCULAR TUMOR. Y. KOYANAGI, Arch. f. Ophth. **134:** 62 (May) 1935.

Leber believed that the retinal detachments which occurred in cases of intra-ocular tumors were caused by a subretinal transudate the source of which was the vessels of the very vascular sarcomas or the tumor cells themselves. Koyanagi reports the anatomic findings in one far advanced case of sarcoma of the choroid, a disease which is very rare around Sendai, Japan. The tumor occupied three fourths of the vitreous cavity; the retina was completely detached. He found certain changes in the cells of the pigmented epithelium which he interprets as signs of active secretion. The secretion consisted of albuminous droplets, which were found in the cells and in the subretinal space. In cases of glioma retinae with retinal detachment the cells of the pigmented epithelium exhibited similar changes. In cases in which the gliomas were largely necrotic, even the pigmented epithelium of the iris showed signs of a secretory process. P. C. KRONFELD.

Trachoma

RECENT ADVANCES IN TRACHOMA. F. H. STEWART, Brit. M. J. 1: 1261 (June 22) 1935.

Present Theories.—There are at present two important theories as to the origin of trachoma:

1. The disease is due to Bacterium granulosis, which was isolated by Noguchi in 1925 from trachoma in North American Indians. He claimed it produced characteristic lesions in chimpanzees and rhesus monkeys. Other investigators have not found it constantly present. It cannot be isolated from the highly infectious conjunctival secretion, and when inoculated into man it does not cause trachoma.

2. Trachoma is due to the Prowazek-Halberstaedter bodies, inclusions in the epithelial cells of trachomatous conjunctivae, discovered in 1906. The early stages of such an inclusion are called initial bodies. These may be free, in which case they cannot be distinguished morphologically from polar-staining bacilli such as Pasteurella avicida; or they may be intracellular, in which case they take various shapes resembling cocci, bacilli, etc. In the fully developed state the inclusion consists of a vacuole filled by a vast number of granules, "the elementary granules," each about 0.25 micron in diameter. With the Giemsa stain, all "initial bodies" and all bacteria stain dark blue while the elementary granules stain purple. The inclusion occurs in from 40 to 100 per cent of cases of trachoma. The elementary granules in the unaggregated state have no typical characteristics and cannot be cultivated. When material from an eye with inclusions is transferred to another eye of man or monkey, inclusions may form in the second eye. In man, following such transference, the formation of inclusions is preceded by a vast num-

ber of initial bodies identical with staphylococci or streptococci, or by an acute Koch-Weeks conjunctivitis, or by an acute conjunctivitis of undetermined nature. In the human cases typical trachoma developed, but, of the inoculated monkeys, only 25 per cent presented inclusions, which sometimes were accompanied by clinical trachoma but more often were not, while clinical trachoma sometimes appeared without inclusions. Filtration experiments gave negative results except in 3 cases.

Summary of Work at Giza, Egypt.—Laboratory work at Giza during the past five years may be summarized as follows: 1. Baboons and grivets were found to be extremely susceptible to Egyptian trachoma in every case, follicles appearing in the whole upper retrotarsal conjunctiva from twenty to thirty days after inoculation.

2. In an examination of conjunctival films in 300 cases, Prowazek-Halberstaedter bodies were not found in pure, uncomplicated trachoma, but only in trachoma complicated by a secondary infection (especially an infection with the Koch-Weeks bacillus or the gonococcus).

3. The so-called initial bodies in such cases are bacteria phagocytosed by epithelial cells. As the bacteria are digested, elementary granules appear in their place.

4. In infected monkeys inclusions were found only in 2 of 26 experiments (and then in very small numbers). Free elementary granules were found only in 4 of the 26 experiments. Monkeys' eyes are relatively free from pathogenic germs and never harbor Koch-Weeks bacilli or gonococci.

5. After a suspension of virulent trachoma secretion has been passed through Berkefeld V candles or gradocol membranes with pore diameters of 0.6 and 0.7 micron, the filtrate is not infective, although it may contain elementary granules.

Hypotheses.—The hypothesis is suggested that the granular virus of trachoma is introduced into the conjunctiva in the bodies of bacteria of several species, the Koch-Weeks bacillus and the gonococcus being the most important in Egypt. These bacteria, acting as intermediate hosts, are phagocytosed to form the Prowazek-Halberstaedter bodies, which are the portals of entry for the elementary granules of the virus. The latter are liberated by the bursting of the inclusions and are dispersed through the conjunctiva. They are then no longer recognizable.

In confirmation of this hypothesis, it may be recalled that in Central Europe and North America the initial bodies resemble, while free, Pasteurella; in Japan, the Malay Islands and in Europe the intracellular forms have the appearance of staphylococci, streptococci, pneumococci, gonococci and Pasteurella; in New York inclusions are formed from Koch-Weeks and other hemophilic bacilli.

The hypothesis explains (1) the resemblance between initial bodies and bacteria, (2) the sudden transition from the former to the very different elementary granules, (3) the finding of the inclusions only in complicated trachoma, (4) their rarity in the relatively aseptic conjunctivae of monkeys, (5) the clinical observation that trachoma follows acute Koch-Weeks ophthalmia in Egyptian villagers (Wilson), (6) the nonfiltrability of the disease agent and (7) its lack of resistance to glycerin.

If this hypothesis is not true, there are two further alternatives: 1. The elementary granules cannot pass a filter when they are in an infective condition. This may be due to a capsule which would double or treble their size, rendering them nonfiltrable, while the nonencapsulated forms are not infective. 2. The elementary granules play no part in the causation of trachoma.

W. F. DUGGAN.

INOCULATION OF THE VIRUS OF INCLUSION BLENNORRHEA INTO TRACHOMATOUS EYES. K. LINDNER, Arch. f. Ophth. **133**: 479 (Feb.) 1935.

Lindner recommends the term "paratrachoma" for all the diseases produced by the genital inclusion virus, namely: (*a*) the inclusion blennorrhea of the new-born, (*b*) the genital diseases of the parents of those babies (genital paratrachoma) and (*c*) inclusion blennorrhea in the adult, transferred to the eye of the adult from either (*a*) or (*b*).

Lindner inoculated the virus of paratrachoma of the new-born into twenty-one trachomatous eyes. In each case the inoculation was done with the consent of the patient after the situation had been thoroughly explained to him. In most of the cases the inoculation produced a very slight inflammation. In some of the cases of trachoma of long standing there were no clinical signs of a new infection. Two patients with folliculosis were treated in the same way. They reacted to the inoculation with an acute conjunctivitis. The conjunctiva of the baboon behaved just like the human conjunctiva in experiments with superinfections. If the first infection was trachoma the effect of the superinfection with paratrachoma was very slight, and vice versa. Thus one disease seems to protect the eye from the other, which speaks for a close relationship between the two types of virus.

P. C. KRONFELD.

Tumors

NEW GROWTH OF THE IRIS IN THE IRIDOCORNEAL ANGLE. P. BAILLIART and R. ROSSANO, Bull. Soc. d'opht. de Paris, April 1935, p. 183.

The authors describe a tumor of the iris observed during a period of three years. The 19 year old patient complained of a discoloration of the right iris. The visual acuity was 7/10 for the right eye and 10/10 for the left. Slit-lamp examination showed a nodule on the anterior surface of the iris between 1 and 3 o'clock. It was irregularly pigmented yellow and over it coursed a few newly formed vessels. On the anterior surface of the lens were a few black pigment deposits. The pupillary reflexes of both eyes were diminished. There was no history or findings of tuberculosis or syphilis. Provocative mercurial treatment caused at first a clouding of the aqueous, then hypopyon formation, but the Wassermann reaction remained negative. Atropine was used locally, and the administration of iodides and mercury by mouth was continued. The media cleared, the tumor became smaller, and visual acuity was enhanced to 10/10. Gonioscopy showed a very small nodule remaining, which appeared calcareous. The case is presented to show the difficulty in making a differential diagnosis between a possible benign gumma or tubercle and a malignant tumor.

L. L. MAYER.

Vision

SLIT-LAMP EXAMINATION OF THE ALTERATIONS OF THE ANTERIOR SEGMENT OF THE EYE AND OF THE VITREOUS IN DIABETIC PATIENTS. G. VILLANI, Ann. di ottal. e clin. ocul. **62:** 881 (Oct.) 1934.

Villani reports slit-lamp observations of the anterior segment of the eye and vitreous in forty-five diabetic patients. Five of the patients were from 20 to 27 years of age; ten, from 38 to 50; fifteen, from 50 to 60, and fifteen, over 60.

Corneal changes were noted in sixteen of these patients. The changes consisted in extensive opacities of the surface and parenchyma, which were disciform and finger-shaped, central or peripheral, with and without cellular precipitates. The opacities were interpreted as scars of an inflammatory process of the cornea, the etiology having nothing to do with diabetes.

The iris changes included atrophy of the stroma with dystrophy of the pigment, which was distributed in the form of diffuse points on the anterior surface of the iris. Four cases of iritis were observed. In two of the cases the condition was acute and complicated with hemorrhage, and in two it was chronic and complicated with numerous posterior synechiae and an exudate in the form of a thin membrane.

After a résumé of the opinions of the various authors who have written on the subject, it is noted that they do not all agree. Some are inclined to believe that the diabetic cataract begins as a cortical cataract, while others believe that it is a nuclear cataract.

Further findings by Villani included deposits of pigment on the anterior capsule of the lens, while in the anterior subcapsular layer of the lens were noted more or less large round vacuoles.

In addition, the following were noted: whitish superficial opacities in the anterior subcapsular layer; thin white cobweb-like equatorial opacities; polymorphic opacities of the anterior cortical substance localized in the zone close to the equator, and more or less large white punctiform opacities distributed in the cortical layer.

The lens changes, which were always bilateral, coincided with the constant changes observed in the lens in experiments on persons with diabetes; these alterations followed the epithelial lesions which were always observed in the hyperglycemic state.

The opacities usually encountered in these diabetic subjects were of the punctate and coronary type of cataract. These changes so found and considered as presenile by Villani have also been encountered in youthful diabetic patients. He concludes that since one finds the same changes in the adult as in the juvenile diabetic patient the cause must be diabetes. He did not find any evident changes of the conjunctiva, aqueous or vitreous.

P. CARELLI.

THE VISUAL ACUITY FOR WHITE OBJECTS SEEN AGAINST A BLACK BACKGROUND. E. KLENOWA, Arch. f. Ophth. **133:** 402 (Feb.) 1935.

This visual acuity was determined by moving two white squares mounted on a black screen apart until they were seen separately. The

visual acuity under these conditions showed a characteristic relation to the intensity of illumination. At first the visual acuity rose with increasing light intensities; then a maximum was reached beyond which the visual acuity dropped. The location of this maximum of visual acuity depended on the state of dark adaptation of the eye. Kravkov concludes from these experiments that visual acuity depends mainly on contrast sensitivity and on irradiation. The combined action of both factors can account for a maximum in the curve depicting the influence of illumination on visual acuity.

P. C. KRONFELD.

Visual Tracts and Fields

INFLUENCE OF LABYRINTHINE STIMULI ON THE VISUAL FIELD. E. PRESSBURGER and I. SOMMER, Arch. f. Ophth. **133:** 352 (Jan.) 1935.

Galvanic current sent transversally through the skull and irrigation of the ear with cold water were used to stimulate the labyrinth. Contractions of the isopter for a 1/300 red target and enlargements of preexisting central scotomas occurred frequently under the influence of those labyrinthine stimuli. The authors consider these visual field defects to be a part of labyrinthine vertigo. Visual field defects also developed in one case in which the labyrinthine vertigo (because of a paralysis of the horizontal conjugate movements) failed to produce nystagmus. In the other cases the contraction of the field usually paralleled the intensity of the nystagmus.

P. C. KRONFELD.

Vitreous

MY FIRST TEN CASES OF ACUTE SPONTANEOUS POSTERIOR DETACHMENT OF THE VITREOUS. T. BAENZIGER, Arch. f. Ophth. **134:** 23 (May) 1935.

During the period from 1887 until 1893 Baenziger saw eight patients all of whom came with the same complaint of an opacity in the vitreous which had developed acutely, often accompanied by photopia. In ten eyes of these eight patients ophthalmoscopic examination revealed a large slightly mobile solid vitreous opacity, usually floating in front of the disk. The sudden onset and the absence of signs of inflammation or hemorrhage suggested strongly that the opacity had been present before but was not perceptible to the patient. Thus it occurred to Baenziger that the most logical explanation for those opacities might be a separation of the posterior hyaloid membrane from the retina. Since 1893 he has seen from ten to fifteen similar cases every year.

P. C. KRONFELD.

Therapeutics

EXPERIENCES WITH GLAUCOSAN. C. HAMBURGER, Brit. J. Ophth. **19:** 455 (Aug.) 1935.

Hamburger first refers to the value of glaucosan (an epinephrine substitute) in the treatment of iritis. The effect obtained, that is, dilation of the pupil, is preferably maintained by atropine or scopolamine, because the effect of glaucosan will persist for only a few hours. Massage near the limbus enhances the mydriatic action.

In iritis glaucomatosa the double effect of reduction of tension (mollification) and mydriasis "is accomplished by no other medicament, only laevo-glaucosan [an epinephrine substitute] will perform it."

Glaucosan has up to the present remained unexcelled in its effects on the iris when synechiae require to be ruptured.

The well known suggestion of Vanna for its use to determine the propriety of an iridectomy in leukoma of the cornea is mentioned.

In glaucoma simplex glaucosan is capable, in cases in which physostigmine or pilocarpine remains ineffective, of bringing about an appreciable reduction of pressure and sensitizing the eye in such a way that miotics again become effective. Although it cannot be determined definitely in which case of glaucoma simplex the eyes will react by increased pressure, nevertheless it can be maintained from experience that it is primarily eyes of a tension of 40 mm. and above which have such a propensity. When operation fails, glaucosan rather than a second operation is indicated. W. ZENTMAYER.

Toxic Amblyopia

SYMMETRIC INCOMPLETE ANNULAR SCOTOMA OF TOBACCO ORIGIN WITHOUT ENLARGEMENT OF THE BLIND SPOT. E. KRIMSKY, Am. J. Ophth. **17**: 722 (Aug.) 1934.

Krimsky reports the case of a 46 year old man who complained of gradually increasing difficulty in reading over a period of four months, this being initiated by flickering. The patient was found to have bilateral partial annular ring scotomas which, in the absence of other apparent cause, were ascribed to tobacco. The scotomas began to disappear when the patient ceased using tobacco. W. S. REESE.

BLINDNESS OCCURRING IN TWO PATIENTS WITH DEMENTIA PARALYTICA UNDER TREATMENT WITH ACETARSONE (STOVARSOL). SALGO, Bull. Soc. d'opht. de Paris, February 1933, p. 75.

1. A patient, aged 47 years, had had the tabetic form of dementia paralytica for ten years. The visual acuity was 9/10 for either eye. After the tenth injection of 1 Gm. of acetarsone blindness ensued. No changes were noted in the fundi. Heroic measures failed to bring back vision.

2. A patient of 40 years had dementia, disorientation and dysarthria. The visual acuity in each eye was 9/10. He received eight injections of 0.5 Gm. and nine injections of 1 Gm. of acetarsone, which were followed by blindness.

The diagnosis of retrobulbar neuritis in these two cases was based on the amaurosis together with the integrity of the fundus and secondary discoloration of the disks. Salgo questions whether the condition was toxic, resulting from the acetarsone, or due to syphilis. Of 120 patients treated at the Asile de Villejuif with acetarsone for syphilis only these 2 were affected by blindness. Salgo recommends an examination of the fundus following each injection, as studies of the visual fields are not obtainable, owing to the patient's mental condition. L. L. MAYER.

BLINDNESS FOLLOWING THE USE OF ACETYLARSAN (P-OXY-ACETYL-AMINO-PHENYLARSINATE OF DIETHYLAMINE). J. S. CHARAMIS, Bull. Soc. d'opht. de Paris, February 1935, p. 77.

Charamis reviews the history of the use of trivalent and pentavalent arsenicals in syphilis and their effects on the eye and its parts. He discusses the theories as to why pentavalent arsenical may cause toxic effects in the optic nerves or release syphilitic toxins which in turn cause the damage.

The case reported is that of a patient of 59 years with syphilis of thirty years' standing. After the second injection of acetylarsan he became completely blind. In spite of extremely active treatment to prevent the continued toxic effects, the blindness remained permanent. A slight deafness which occurred with the blindness entirely disappeared. Charamis is inclined to believe that the dire effects of the drug are due to the many complications of syphilis, such as nephritis and cardiovasculitis, which this patient showed.

An interesting discussion of this and other papers on the same subject follows.

L. L. MAYER.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

October 1935

J. MILTON GRISCOM, M.D., *Chairman*

ALEXANDER G. FEWELL, M.D., *Clerk*

MULTILOCULAR CYST OF THE IRIS. DR. CHARLES A. RANKIN.

The case reported was that of an implantation cyst occupying the anterior chamber of the eye between 8:30 and 10 o'clock, with two smaller cysts extending from the main cyst out into and involving the pupillary space. There was a history of injury occurring two years previously.

CYSTLIKE ECTROPION UVEAE. DR. ALFRED COWAN.

Two sisters and two children of one of them, four in all, exhibited congenital cysts and flocculi of the pigment layer at the pupillary border.

DISCUSSION

DR. T. B. HOLLOWAY: About twenty years ago a Jew with bilateral glaucoma presented in one eye a pigmented mass projecting beyond the pupillary border, apparently pushing in between the lens and the iris. Later this mass disappeared only to recur at a subsequent date. As is now well known, such cysts may form in the pigment layer of the iris and ciliary body in connection with glaucoma or after inflammation of these structures.

Dr. Rankin referred to cysts of the iris in patients with diabetes. Every experienced operator has seen the escape of an inky fluid into the anterior chamber when performing an iridectomy in certain cases of diabetes. But this may occur in the absence of active diabetes, as was shown several years ago in two patients whose urine was free from sugar and whose blood sugar was well within normal limits.

DR. ALFRED COWAN: Regarding a free cyst in the anterior chamber—it is supposed to be a cyst that previously was attached by a pedicle, which has been torn loose. But nowhere have I seen—nor does there seem to be a record of a case—a free cyst in the presence of attached cysts.

DINITROPHENOL POISONING WITH CATARACT. DR. EDMUND B. SPAETH.

A case was presented in which obesity had been corrected through the use of dinitrophenol. After eleven months of medication cataracts

developed, and within six weeks almost complete liquefaction of the cortex had occurred. The patient at the end of the medication began to have clinical and serologic symptoms suggestive of parathyroid tetany.

RAPIDLY DEVELOPING CATARACTS FOLLOWING THE USE OF DINITROPHENOL. DR. WILLARD G. MENGEL, Camden, N. J.

In a woman, aged 33, bilateral cataracts developed rapidly, following the use of dinitrophenol. At the first examination on June 3, 1935, by means of the slit lamp, about 30 or 40 small dotlike opacities were detected in the anterior cortex of the lens, beneath the capsule; otherwise both eyes were normal. The vision became more blurred each week as the opacities increased in number. In six weeks there was considerable swelling of each lens, and the vision in each eye was reduced to 6/21. Eight weeks following the initial examination the vision was reduced to perception of light. The intra-ocular tension was normal.

The laboratory investigations yielded negative results. Determination of the blood sugar showed 85.8 mg., and that of blood calcium, 10 mg., per hundred cubic centimeters. The Wassermann reaction of the blood was negative. The roentgenologic examinations of the teeth, sinuses and sella turcica revealed nothing pathologic.

Operation was performed on the right eye September 9, and deferred on the left for a few weeks. By linear extraction the soft lens material was completely removed without difficulty. The convalescence following the operation was rapid and without complication. Permanent glasses were not ordered, but trial refraction showed the vision to be 6/12 plus.

A calculation of the amount of sodium alpha-dinitrophenol used showed that approximately 118,000 mg. was taken during the fifteen month period from March 1934 to June 1935. During this period there was a reduction in weight from 217 to 170 pounds (98.5 to 77.2 Kg.), a total loss of 47 pounds (21.3 Kg.).

The striking features were the rapid development of identical cataracts simultaneously in the two eyes, the comparative youth of the patient and the absence of diabetes and trauma. Except for the use of dinitrophenol no other etiologic factors were discovered. From this apparent association of the dinitrophenol therapy with the formation of cataract it seems that the use of the drug was definitely contraindicated.

DISCUSSION

DR. EDWARD STEINFIELD: There is little doubt that dinitrophenol was the cause of the cataracts in both the patients presented tonight. I shall limit my discussion to the disturbance in the metabolism of calcium noted in Dr. Spaeth's patient. The fact that mild tetany occurred when the calcium fell to 7 mg. per hundred cubic centimeters (the critical level) simply indicates a hypocalcemic tetany, though the proportionate lowering of the level of phosphorus is not characteristic of parathyroid deficiency. Tetany may appear at higher levels of calcium if there is a rapid drop, as in parathyroidectomy. On the contrary, low figures may be noted in nephrosis owing to loss of the components

bound with protein and yet without tetany. The prompt recovery after administration of parathyroid extract is, however, suggestive. Whether disturbances in the metabolism of calcium will be found in other cases of cataract caused by dinitrophenol, I do not know. The possibility of associated nutritional disturbances due to a rigid or unbalanced diet should be kept in mind. It appears advisable to investigate future cases of this character along the lines suggested by Dr. Spaeth's paper.

DR. LOUIS LEHRFELD: I should like to place on record a case similar to that reported by Dr. Mengel: A nurse, aged 27, with a history of failing vision, consulted me with the idea of having her glasses corrected. Routine examination revealed small punctate dots on the posterior cortex of each lens. At first I thought they were congenital and paid no particular attention to them. When she returned with her new glasses I was amazed to find the vision was less than before and that the punctate dots were now striate opacities.

The opacities increased rapidly from day to day until, forty days after the first examination, she had fully mature cataracts and vision in both eyes was reduced to perception of light. Her employer informed me that one year previously she had taken a course of dinitrophenol therapy. At that time she began to have definite loss of hearing, which recovered after treatment. Because of the patient's attitude of secrecy in relation to her history, her employer and I believe that she had taken dinitrophenol just prior to her recent ocular trouble. The cataracts were removed by simple extraction, and with glasses the vision was corrected to 5/5 in each eye.

A REVIEW OF INFRA-RED PHOTOGRAPHY: WITH REFERENCE TO ITS VALUE IN OPHTHALMOLOGY. DR. JACOB B. FELDMAN.

This article will appear in a later issue of the ARCHIVES OF OPHTHALMOLOGY.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Nov. 8, 1935

MR. RANSOM PICKARD, C.B., M.S., *President*

FUCHS' EPITHELIAL DYSTROPHY. MR. HUMPHREY NEAME.

A woman, aged 60, was first seen in September 1933 at Moorfields, complaining of a six months' weakness of the sight of the left eye. At that time the corrected vision was 6/9 in the right eye and 1/60 in the left eye. The slit lamp showed changes in the posterior reflecting zone and epithelial edema over the cornea. The tension was normal, the pupils were equal and active, and there was a deep anterior chamber. She had been treated with thyroid tablets and 5 per cent diamine drops. Last May the microscope and the slit lamp revealed a coppery reflex in the posterior reflecting zone of the right eye.

DISCUSSION

MR. J. H. DOGGART: The epithelial edema was very marked; there were many vacuoles and changes in the substantia propria, so that inspection was difficult.

The right eye showed a much earlier stage of dystrophy. This disease usually starts in one eye, but there is not usually so much difference between the two eyes as in this case. Characteristic changes occurred in the endothelial cells by means of which they became amorphous, so that the picture was no longer that of a clearcut mosaic; holes, consisting of localized protrusions of Descemet's layer into the anterior chamber, also occurred in the epithelium.

MACULAR SARCOMA. MR. HUMPHREY NEAME.

Two months ago the left eye of a woman showed in the macular area an ill defined rounded prominence of slightly paler color than its surroundings. There was a small hemorrhage near a transverse superior temporal vessel. During the last two weeks the pigmented area has definitely increased in size and now stretches across the lower third of the swelling. The hemorrhage to the temporal and inferior temporal side and beneath the level of retinal vessels has definitely increased.

DISCUSSION

MR. RANSOM PICKARD: It is my feeling that this is more likely a solid growth. In the few cases of cysts in that situation which I have seen they have not been accompanied by much hemorrhage.

MR. MONTAGUE HINE: I think it is more likely a hemorrhagic exudate into the choroid associated with hemorrhages.

ANGIOSARCOMA OF THE CHOROID. MR. J. D. CARDELL.

A little girl, aged 7, in May 1934 presented a cataractous and tremulous right lens and was suspected to have a tumor in the right eye. The patient was seen again in May 1935, when a fungating tumor was seen to be protruding from the front of the right eye. An excision was performed three days later and an extension of the growth found along the sheath of the optic nerve. The patient was treated with roentgen rays of high voltage, but had a cerebral seizure at the end of September and died four hours later.

The eyeball was greatly distended and quite filled with the growth, and there was extra-ocular extension. Bands of fibrous tissue extended from the deep surfaces of the sclera into the substance of the growth, and fine extensions from these bands passed from individual groups of cells and formed a fibrous reticulum for the whole neoplasm. The cells of the growth were typically sarcomatous, large and fusiform, and each with an oval nucleus. Two structures composed the growth: a cavernous angiosarcoma in the area bordering on the original uveal tract, and elsewhere a fibrosarcoma with an unusually well marked reticulum. There was no trace of melanotic or hematogenous pigmentation, an observation which appears to have been made in most of the reported cases of angiosarcoma of the eye. Malignant invasion of the cornea occurred by way of the new vessels present there. The inter-

vaginal sheath of the optic nerve was filled by growth, though neither the sheaths nor the nerve fiber bundles were infiltrated.

A Congenital Retinal Anomaly. Mr. Frank Law and Miss Ida Mann.

Mr. Frank Law: An eye of a girl, aged 6, had been excised under the suspicion that it contained a glioma. A massive exudate was present over the ciliary body with a curious distribution of the growth. The parents and the brother and sister of the patient were healthy. The mother had not been exposed to x-rays during pregnancy. No external abnormality was noted. Section was made in the sagittal plane on the nasal side, just clear of the lens. There was a thick cord, 2 mm. in diameter, running from the disk to an attachment at the extreme temporal equator of the clear lens in the horizontal meridian. From the anterior end of this cordlike structure there passed two expansions to be attached to the structures in the region of the ciliary body. These were reflected backward on the inner tunic of the globe in a fan-shaped manner as far as the ora serrata, fading into the inner tunic. The upper expansion was further reinforced by a web of embryonic tissue, found to be a sort of retinal mesentery. This extended the whole length of the cord. The cord was very vascular and very firm to the touch, and was connected with the disk, lens and web. Along the line of insertion of the web were seen dense white bodies which seemed to be formed by calcareous depositions in the tissues.

The right eye of a boy, aged 22 months, was excised under suspicion of glioma. He had been noticed to have a right convergent strabismus for four months. The anterior chamber was found to be shallow. The pupil was sluggish to light. A localized opacity was present in the outer and lower part of the lens, and an indefinite gray raised area, bandshaped, in the vitreous, with what seemed to be normal retina above and below it. A few weeks before the excision, the vision of the right eye was apparently subnormal; when the eye was excised, a grayish band was found across the fundus, roughly similar in appearance to that in the right eye.

Miss Ida Mann: I feel indebted to Mr. Law for having made clear a condition which has been puzzling me for years. I have found now eight cases of a similar abnormality, and I shall try to suggest the embryonic mechanism which is responsible. With the first case which I saw I did not recognize or realize that it was one of a group of similar cases, but I reported the abnormality as an isolated anomaly. The next example I saw in both eyes in a baby at Moorfields; I have also seen it in a girl aged 9, and have received a report in another case from Mr. Somerville Martin. I am also familiar with the case recently described by Stella Ancona in the *Netherland Journal of Ophthalmology,* and have recently heard from Professor Were that he is publishing a report of further cases this month.

With regard to the possible embryology of such cases, it seems to me that the presence of some trace of the hyaloid artery in the region of the fold in most of the cases may have some bearing. I advance, as a tentative explanation, the possibility that adhesion of the primary (vascular) vitreous to the inner layer of the optic cup in one meridian

may be the determining factor. If the secondary vitreous failed to form along this meridian, a fold of retina would be pulled up into the interior of the eye. The cause of the adhesion must remain unknown. The varying situation of the fold shows that it has nothing to do with the fetal fissure, and the fact that it is sometimes bilateral and symmetrical seems to exclude inflammation or fortuitous organized hemorrhage as the cause. At present it appears to be a rare anomaly, but since in most of the cases the anomaly was at first suspected of being glioma and later was diagnosed as pseudoglioma, it is possible that it is not rare but has merely escaped recognition.

DISCUSSION

MR. A. E. ILES: A child appeared at the Bristol Eye Hospital eighteen months ago with a left convergent squint. Retinoscopy showed a retinal band running from the disk directly outward from the margin to the position of 2:30 o'clock.

FUCHS' EPITHELIAL DYSTROPHY. MR. J. H. DOGGART.

This is a rare bilateral disease, slowly progressive, of unknown causation, which affects elderly people, especially women, and for which no cure has yet been discovered.

The first case is that of a woman, aged 70, whom I saw for the first time some years ago. She has been coming up every few months for inspection, as she had a hypopyon ulcer in the right eye, the cornea of which is now more than half covered by a dense vascular scar. The left cornea is hazy but shows no vessels. There is no history of trauma or evidence of inflammation other than mild conjunctivitis. The tension of the eye is normal. The corneal endothelium does not exist in any recognizable form. The cell mosaic normally covering the back of the cornea has been replaced by a layer of granules resembling red pepper.

The second patient, aged 72, for some time had noticed that her eyes seemed "sticky." She had been able to see better in the evening than in the morning. Her fields of vision and the fundi were normal. There was a faint central haze on each cornea; she had marked edema of the epithelium in both eyes, and there were a few irregular striae in various layers of the substantia propria. In each cornea the sensation was diminished. She began to notice the halos which are a sequel to epithelial edema.

Fuchs, in his Bowman Lecture of 1902, spoke of a corneal condition in elderly people which could simulate glaucoma and speculated on the possibility that this condition might be due to some disease of the endothelium. In 1910 Fuchs published a paper with carefully worked out clinical observations on thirteen cases of the condition, which he called "epithelial dystrophy of the cornea." Nine of the cases were in elderly women; in three of these cases chronic glaucoma developed.

Intermittent conjunctivitis is common. As time goes on there is an increasing tendency for these blebs to rupture. Hence there is considerable risk of infection, and ulceration of the cornea has been recorded more than once. The loose attachment of epithelium calls for great care in the application of the tonometer. The association of the condition with myxedema has led to the idea that administration of

thyroid may benefit, but I think there has never been success from this treatment. Epithelial dystrophy has been described in association with myotonia atrophica, and this is further evidence that the state of the epithelium is bound up in the general metabolic state. Many drugs and physical means have been tried without success.

DISCUSSION

MR. GRAY CLEGG: In a paper written in 1915 I described five cases of corneal dystrophy, three of them in sisters. In the case of an old gentleman, I confused the condition with glaucoma and did a trephining. In one case the epithelial condition came on after the trephining, the cornea having previously been clear.

MR. T. HARRISON BUTLER: The silvery appearance at the back of the cornea is not so rare if looked for, especially when this condition is mild. From the medicolegal point of view it is important to differentiate cases due to accident from those which Mr. Doggart has described.

DR. A. J. BALLANTYNE: Having seen about thirty cases of dystrophy, I feel that its frequency is not more than 1 in 10,000 to 14,000.

MR. FRANK JULER: I should like to ask what is considered to be at the bottom of the bronze appearance of the cornea. Graves thought it was due to a scattering of light by reflection from minute protrusions of Descemet's membrane.

MR. J. H. DOGGART: I think Mr. Gray Clegg's cases reported in his paper were instances not of this disease but of bullous keratitis. There is no association of this condition with trauma, though I agree that in association with trauma there can be epithelial changes ranging from fine edema to that of a grossly bullous state. If there were an association of the condition under discussion with trauma, two thirds of the cases would not be likely to be met with in women, as is the case. This powdered bronze appearance represents something different from the ordinary pigment granules that result from the scattering of pigment in the iris in old people.

FRENCH OPHTHALMOLOGIC SOCIETY

Forty-Eighth Annual Meeting, Paris, May 13-17, 1935

ABRIDGED TRANSLATION FROM AUTOREFERATA OF READERS OF PAPERS BY PERCY FRIEDENBERG, M.D., NEW YORK

(*Continued from vol. 14, p. 1053*)

DISCUSSION OF PAPER ON CAPSULOLENTICULAR EXTRACTION OF CATARACT READ BY DR. DE SAINT-MARTIN, TOULOUSE

DR. M. E. KALT, Paris: It was in 1908, twenty-seven years ago, that for the first time and not without emotion I extracted a cataractous lens with the forceps which bears my name and presented the opaque lens at the tip of the instrument to my assistants at Quinze-Vingts.

This forceps which neither pricks nor cuts is intended to allow the pulling out of the lens entire in order to avoid secondary cataract. Operative experience proves that the zonule of Zinn is sufficiently brittle to permit this maneuver without danger to the eye. I soon convinced myself that a recent white cataract with a thin, delicate capsule does not lend itself to this form of operation. During the first few years in which I practiced this new form of extraction I was able to obtain only 10 per cent total extractions. The operative result was magnificent. I operated with the eye under local cocaine anesthesia, and the procedure, while apparently dangerous, was most impressive. Besides total extraction, I practiced the removal of a large flap of the anterior capsule and thus greatly reduced the number of secondary cataracts. In 1925 I extracted the lens (intact) in 25 per cent of the cases in which I operated; in 1929, in 33 per cent; in 1934, in 55 per cent, and at present I succeed in 60 per cent. Corneal suture, practiced as a routine since 1894, has proved indispensable. Without it, I should never have dared to keep on in this path. The transverse branch of the T, which is very short, allows a proper coaptation of the edges of the wound. Afraid to trust to foreign aid, I operated without an assistant, alone, separating the lids with the fingers of my left hand. The invention, in 1914, of a suture passed through the superior rectus muscle gave me more complete control of the globe.

I found that the torn capsule, when not extracted completely, could become wedged or entangled in the lips of the operative incision. Accordingly, I preserved the iris intact and made only a small peripheral iridectomy as a final step. For this purpose I used a small sharp hook in preference to an iris forceps or incision of the iris with a de Wecker scissors. The securing of orbital anesthesia by injection of procaine hydrochloride proved to be an immense step forward: Operation on the fixed globe lost much of its intimidating character. The aid of an assistant became permissible. He was charged with retracting the lower lid by means of a Desmarres hook and keeping stretched the thread through the superior rectus muscle. I was thus able to extract the lens in the manner taught me by my former masters, making pressure on the lower part of the globe with a bent curet. That is my procedure at present.

Just a word on the subject of the forceps. It should not prick or cut, and the branches should be parallel and not form a V. Many badly made examples have been put on the market and have got into circulation. This explains certain disappointments.

To sum up, I operate nearly as I did twenty-five years ago but with more confidence and boldness. Modifications of the procedure, which have been made from several sides, have not made any essential change. The operation has been accepted throughout the entire world, and the skeptical smiles which greeted my first communications have disappeared. Let me say I am satisfied with this.

Dr. A. Terson, Paris: The term "complete (*intégrale*) extraction" best characterizes a procedure in which the two principal modes are vacuo-avulsion with negative air-pressure and forci-avulsion with the forceps —the latter procedure was originated by Daviel long ago. At present, palpebral akinesia and orbital anesthesia allow one to perform total

extraction more generally, except in young children. However, in cases of sufficiently mature cataract, extraction of the lens after laceration of the anterior capsule with the forceps exposes to fewer complications and failures, gives excellent visual results, and is by no means about to be abandoned as an operative measure.

DEMONSTRATION OF APPARATUS FOR EXAMINATION OF THE EYE. DR. LÓPEZ-ENRÍQUEZ, Madrid, Spain.

The demonstration was followed by an official visit to the exposition and display of instruments and their presentation by the exhibitors.

Third Session, Wednesday, May 15

OCULAR COMPLICATIONS AND SYMPTOMS OF "FIÈVRE EXANTHÉMATIQUE." DR. G.-E. JAYLE and DR. P. MASTIER, Marseilles.

The classic signs of this disease, which has also been termed "exanthematic coast fever" (*fièvre exanthématique du littoral*), are initial conjunctivitis, which may be followed a little later by mild keratitis, and inoculation conjunctivitis through ocular projection of the virus of the disease. Besides these reactions it appears that a true complication may occur in the form of an exanthematic neuroretinitis, characterized by involvement of the vessels limited to a single branch of the central artery of the retina, with a quadrantic defect in the visual field. This may come on while the disease is progressing or later when it is coming to a standstill. Four cases have been observed at the clinic for diseases of the eye at the Hôtel-Dieu in Marseilles within the last few years.

THE PHYSIOPATHOLOGY OF RETINITIS (Lantern Slides). DR. A. FRITZ, Mons, Belgium.

Direct observation of the retinal circulation of the blood in certain stages of nephritic or hypertensive retinitis showed a condition which does not square with Volhard's theory that ischemia is the main etiologic or pathomechanical factor. A condition of hypertension with atonic dilatation at all levels of the retinal vascular tree up to and including that of the capillaries was evidenced by the broad arteries with markedly pulsating walls and a low Fritz index, with tension raised to a point near that of the humeral artery. Further corroborative evidence was given by the marked capillarity of the disk and its coloration thereby and finally by the dilated veins, which were generally the seat of high tension and marked pulsation varying markedly between venous minima and maxima which were widely separated. The question suggests itself as to whether retinitis of nephritic hypertension does not mark the arrival of the hypertensive process at the level of the capillaries by putting into play a progressive relaxation of the arteries, the arterioles, and finally of the precapillaries. The clinical course of true cases in which unilateral retinitis is present may, in fact, show that hypertension in the capillaries is more marked in the affected eye and that one may obtain retrogression of the retinitis by causing a lowering of general blood pressure with consequent diminution of retinal arterial

tension. The therapeutic successes obtained by lumbar puncture after Magitot may well be due in the same way, at least in part, to a lessening of the venous stasis and the consequent disembarrassment and relief of the capillary circulation of the retina.

RETINAL ANGIOSCOPY AND EARLY DIAGNOSIS OF CARDIOVASCULAR DISEASE. DR. JEAN GALLOIS and DR. R. GIROUX, Paris.

Observations were made on young subjects: 1. Increase of caliber and broadening of the arteries were noted in hypotonic states and with arterial hypotension. If retinal arterial tension is found to be raised, the circulatory system should be examined, special attention being paid to the aorta. Four cases of aortic dilatation were detected in this way, all in young subjects. 2. Retinal arteritis, which is so common in aged arteriosclerotic patients, is rare in children, adolescents and young adults, and its presence should always make one think of lesions, slight but widespread, in the peripheral arteries. Attention to this point has allowed the detection of an anatomic substratum in subjects affected with spasms of the peripheral vessels or with angina pectoris, and in two patients 28 and 17 years of age, respectively, it has led to the discovery of aortic lesions which had not been recognized.

THE OCULAR PULSE (Lantern Slides). DR. P. BAILLIART, Paris.

No abstract was submitted.

NEURO-EPITHELIOMA OF THE RETINA. DR. R. PERRIN and DR. NOEL, Lyons.

No abstract was submitted.

RECENT CYTOLOGIC STUDIES OF PATHOLOGIC VITREOUS. DR. LÓPEZ-ENRÍQUEZ, Madrid, Spain. (From the University Ophthalmologic Clinic [Professor Marquez] and the Laboratory of Normal and Pathologic Anatomy [Director: del Rio-Hortega].)

[Projections were made of photomicrographs showing Hortega cells which had migrated from the retina.] Stress is laid on the morphologic alterations undergone by these elements as they reach the vitreous. Furthermore, this observation was made by me as far back as 1931 and is of particular interest so far as it concerns the cytology of the pathologic vitreous, since by far the greater part of the cellular elements found there are nothing more or less than Hortega cells from the retina. I also report the continuation of my studies on optical magnification. Investigations which were made practically possible by the use of improved and more precise instruments oblige me to modify some interesting points of my theory and to state definitely that in the case of the loupe—when the observation is made with parallel rays—the magnification is always the same and remains unaltered for each system, whatever the distance between the eye and the condensing lens, even when the latter is in actual contact with the cornea. Furthermore, the accepted and traditional theory of (subjective) optical magnification is false. The projection of the retinal images takes place in the same plane as the object—real or virtual—for which the eye is focused.

THE PATHOLOGIC HISTOLOGY AND PATHOGENESIS OF RETINAL DETACHMENT (Lantern Slides). DR. J. MAWAS, Paris.

No abstract was submitted.

NOTE ON THE MEDICAL PROBLEM OF IDIOPATHIC DETACHMENT OF THE RETINA. DR. JEAN GALLOIS, Paris.

The numerous disturbances, local and general, observed in patients with retinal detachment are reviewed. Attention is called to studies which tend to show that this pathologic process, which is not due to a single cause, does not take place except in a predisposed eye and a predisposed organism. The etiologic conception that a retinal tear is the one and only invariable factor in the causation of detachment leads logically to the incorrect belief that obturation of the tear is a radical therapy which at once and completely does away with the cause of the trouble. This fails to take into consideration all of the clinical signs. Beyond doubt one must back up operative procedures, however necessary, by appropriate medical treatment if one is to avoid recurrences and preserve the other eye.

RETINAL TEARS WITHOUT DETACHMENT. DR. P. GUILLOT, Marseilles.

Observations suggest that there are certain signs on which one may base a prognosis as to whether a tear will be followed by detachment and whether a limited detachment will spread and become generalized. This is based on the presence of a delimiting, barrage-like choroiditis; on the fixed aspect of the detached retina which stands off perpendicularly (sic) from the choroid like a swelling sail, and on the worn and "hammered" (*martelé*) appearance of the retina at points where it is going to become detached.

SOME QUESTIONS ON DETACHMENT: 1. THE IMPORTANCE OF THE ANAMNESIS IN THE SEARCH FOR TEARS. 2. FALSE TEARS (*Pseudo-déchirures*). 3. STUDY OF PHOSPHENES. DR. LÓPEZ-ENRÍQUEZ, Madrid, Spain.

No abstract was submitted.

RETINAL DETACHMENT AND POSTOPERATIVE HEMORRHAGE. MME. NOELLE CHOMÉ-BERCIOUX, Lausanne, Switzerland.

Two recent spontaneous detachments, one of one month's, the other of eight days' standing, were operated on by diathermocoagulation and by diathermocoagulation and thermocautery, respectively. Twelve days after operation in one case, and nine days after, in the other, a massive hemorrhage into the vitreous occurred, rendering ophthalmoscopic examination futile. In spite of this there was complete recovery in the later clinical course. In a third case, one of detachment following extraction of cataract, there was an intra-ocular hemorrhage on the thirteenth day after operation for reattachment. This was completely absorbed in the course of three months, and the final result was recovery with a return of vision to the optimum noted before the detachment. Accordingly, it seems that one might be somewhat less guarded in prognosis than one usually is in regard to these hemorrhages.

EXPERIMENTAL STUDY OF EYE AND HEAD MOVEMENTS TRANSMITTED TO DETACHED RETINA. DR. TOULANT, Algiers, Algeria.

No abstract was submitted.

PROGNOSIS IN THE SURGICAL TREATMENT OF RETINAL DETACHMENT (Lantern Slides). DR. H. ARRUGA, Barcelona, Spain.

No abstract was submitted.

THE OPERATIVE TREATMENT OF DETACHMENT OF THE RETINA. DR. L. WEEKERS, Liége, Belgium.

In order to get the detached retina to become "soldered" to the shell of the globe, one must try to get the intervention of the choroid and of the episcleral tissues. It is not possible to state which of these factors is the more important. The best technic for the production of cicatricial episclerochoroidal reactions having all the requisite qualities consists in thermo-acupunture with deferred perforation. By means of the galvanocautery one may make a series of little creases in the sclerotic to provoke foci of inflammatory reaction in the choroid. At a later stage, by means of a cataract knife, these foci are pierced to allow penetration by the episclera. The advantages of this procedure are appreciable and evident, namely: the favorable ophthalmoscopic appearance of the cicatrices, which are pigmented; the rapidity with which the cicatrization is completed in the course of a few days; the minimization of any extension of inflammatory reaction or cicatrical changes to the neighboring retina; the firm cicatrization, as demonstrated and followed up by histologic study, and finally the ease of execution of the procedure, which is by no means a negligible factor.

Fourth Session, Wednesday, May 15: Joint Session with the Ophthalmological Society of Paris.

PRESENTATION OF A PYROMETRIC CONTROLLER WITH VARIABLE ZERO FOR OCULAR DIATHERMY. DR. P. PESME, Bordeaux.

An apparatus is described which is designed to regulate the temperature produced in tissues in contact with an active electrode. It consists essentially of a thermo-electric couple which acts both as a surgical instrument and as a gage of the heat produced. This first couple is connected with a galvanometer graduated in degrees of temperature. At the start the galvanometer can be regulated with reference to the surrounding temperature by means of a set screw or button on the front face. A flat electrode permits contact heating as in transscleral diathermy, and a needle-pointed electrode is used for coagulating micropunctures. The apparatus is simple, accurate, reliable and portable. It is practical in that it can be fitted to any diathermy central.

PERSONAL EXPERIENCE WITH TREATMENT OF RETINAL DETACHMENT BY MEANS OF DIATHERMOCOAGULATION. DR. P. AVIŽONIS, Kovno, Lithuania.

The procedure described consists in applying the current through an olive-pointed, i. e., blunt, electrode to the detachment in the immediate

neighborhood of the tear, if one has been localized. At the end of this maneuver a number of perforations are made by pricking the coagulated areas with a needle-pointed electrode. Forty-nine operations were performed on thirty-three eyes of thirty-two patients. In fifteen cases there was complete cure with obliteration of the tear and definite reattachment of the retina; in three instances there was definite improvement with a small area of retina still remaining unattached; in two other cases there was satisfactory improvement; in thirteen cases the detachment remained unimproved. Twelve patients had a detachment in the second (*?dernier*) eye with recovery in six and improvement in two cases. As complications, I have noted iridocyclitis, not severe, in one case and serious and obstinate vitreous opacities, which, however disappeared, little by little, in three.

BASIC PRINCIPLES OF TREATMENT OF RETINAL DETACHMENT. DR. GILBERT SOURDILLE, Nantes.

No abstract was submitted.

DIATHERMIC DIAPHANOSCOPE. DR. M. STRAMPELLI, Rome, Italy.

In order to apply the diathermic coagulating electrode exactly at the point of localization of the retinal tear, I have thought out and had made by the Zeiss firm a novel apparatus which consists in a transilluminator and diathermic electrode combined. This gives a cone of transscleral illumination the point of which functions as a diaphanoscope and as a diascleral diathermic electrode at one and the same time. This allows one to control, by direct observation through the pupil during operation, the position of the electrode in relation to the retinal tear and also to observe directly and control the coagulation taking place in the choroid, all of which is of decided practical value in the operation for detachment of the retina.

REMARKS ON ONE HUNDRED CASES OF RETINAL DETACHMENT TREATED BY DIATHERMOCOAGULATION. MME. SCHIFF-WERTHEIMER and DR. BAILLIART, Paris.

No abstract was submitted.

DIATHERMOCOAGULATION IN THE TREATMENT OF RETINAL DETACHMENT. DR. GUGLIANETTI, Turin, Italy.

No abstract was submitted.

COMPARATIVE VALUE AND RESULTS OF THE VARIOUS OPERATIVE PROCEDURES FOR THE OBLITERATION OF RETINAL TEARS. DR. P. VEIL and DR. M.-A. DOLLFUS, Paris.

No abstract was submitted.

TREATMENT OF RETINAL DETACHMENT BY MEANS OF THE NEWER OPERATIVE PROCEDURES. DR. GONIN, Lausanne, Switzerland.

No abstract was submitted.

IMPROVEMENTS IN THE TECHNIC OF PYROMETRIC DIATHERMOCOAGULATION IN THE TREATMENT OF RETINAL DETACHMENT. DR. L. COPPEZ, Brussels, Belgium.

The pyrometric electrode which I presented for the first time in 1932 has been modified by several mechanical alterations that improve it by making manipulation of it easier and widening its field of action. This new model makes operation possible in cases which would otherwise be considered unfavorable, with a maximum prospect of success and with absolute security. (The author reviews in detail the advantages and disadvantages of his technic of mild superficial pyrometric diathermocoagulation over a large area, on the one hand, and the procedure of micropuncture on the other. He concludes by demonstrating once again that a surface diathermocoagulation cannot be conceived otherwise than as under control of the pyrometric electrode.) The authors who at the present day persist in depending on the intensity of the current as an indicator of the degree of coagulating energy misunderstand the laws which rule high frequency currents.

TWO YEARS OF PRACTICE WITH DIATHERMOCOAGULATION. DR. VILLARD, Montpellier.

No abstract was submitted.

DIATHERMY AND THERMOPUNCTURE IN RETINAL DETACHMENT (PRESENTATION OF PATIENTS). DR. A. POLACK, Paris.

No abstract was submitted.

SUTURE OF THE RETINA: DESCRIPTION OF THE PROCEDURE DEVISED FOR THE PURPOSE OF FASTENING THE DETACHED RETINA BY MEANS OF SUTURES. DR. R. RUBBRECHT, Bruges, Belgium.

No abstract was submitted.

Fifth Session, Thursday, May 16

STATISTICS ON INJURIES TO EYES IN THE COAL INDUSTRY. DR. J. BADOT, Jumet, Belgium.

Analysis of several thousand cases of ocular traumatism in mining and transporting coal shows 30 per cent due to blunt violence (*traumatismes fermés*) and 70 per cent due to wounds and foreign bodies (*traumatismes ouverts*), whereas in other industries, particularly the metallurgic trades, injuries of the second type are much more common and the percentage is about 85 to 90. Among the "closed" injuries, the most practical importance attaches to contusions of the globe with intra-ocular hemorrhages or hematomas or both. Foreign bodies in, and wounds of, the cornea are the most frequent and common forms of "open" traumatisms.

OCULOFOCAL DISEASE. DR. KAPUSCINSKI, Posen, Poland.

No abstract was submitted.

THE GROENBLAD-STRANDBERG SYNDROME: ITS RELATIONS TO OTHER INVOLVEMENTS OF THE MESENCHYME. DR. A. FRANCESCHETTI and DR. E. ROULET, Geneva, Switzerland.

No abstract was submitted.

THREE CASES OF ANOPHTHALMIA (CLINICAL OBSERVATON). DR. MOUTINHO, Lisbon, Portugal.

Three cases of apparent anophthalmia or, better, clinical anophthalmia, since the patients are actually devoid of eyes, constitute a remarkable and unusual series. At my clinic, in which a hospital and an outpatient department are combined with my private clinic, upward of 102,400 patients have been treated since 1902. Still, it was not until December 1933 that the first case of congenital anophthalmia was seen at the hospital. This condition was associated with colobomatous cysts of the lower lids. The other two cases were seen soon after at the private clinic, in December 1934 and February 1935, respectively. All three children showed markedly shortened palpebral fissures, about from 8 to 10 mm. long, with marked invagination of the lids. The lower lids presented fluctuating protruding cysts of a bluish color, each about the size of a small almond. The orbits were empty. Of these three cases, only the last harmonizes with accepted ideas and the views of the great majority of writers on the importance of consanguinity and heredity in the etiology of this congenital malformation. In the other two cases there were absolutely no such factors as hereditary antecedents, consanguinity, disease or other disturbances during the pregnancy. These three children all came to term normally, were delivered normally and show no somatic anomaly other than the ocular condition noted. From the etiologic point of view the cases are most exceptional.

TWO NEW CASES OF BLINDNESS DUE TO ACETYLARSAN. DR. TOULANT and DR. MEDINGER, Algiers, Algeria.

No abstract was submitted.

OPTIC NEURITIS OF SPHENOID SINUS ORIGIN AS DOCUMENTED HISTOLOGICALLY. DR. G. WORMS, Paris.

The rôle of foci of infection in the sphenoid sinus in cases of optic neuritis is still a subject of discussion. Accordingly, the details of a case in which the causal relation is attested by irrefutable anatomic observations deserves to be received with more than usual interest. The symptom complex consisted in dilatation of the sella turcica, bilateral atrophy of the optic nerve, and the adiposogenital syndrome, and there were other pathognomonic indications which seemed to impose the diagnosis of tumor of the hypophysis. The patient succumbed to an acute streptococcic meningitis, and histologic study of the globe, optic nerves and sinuses promptly threw light on the origin of the ocular disturbance. The atrophy of the optic nerve, which had come on rapidly, was not due to compression of the nerve by a deformation of the hypophyseal region but to optic neuritis of sphenoidal origin. The marked inflammatory thickening of the sheaths of the optic nerve where

they were in contact with the infected sinuses showed this beyond a doubt. As often happens, the posterior sinuses, which were filled (*habités*) with polyps and pus, had remained silent for a long time, not giving any sign of the presence of these abnormalities by disturbances of a rhinologic nature. The sinus infection was communicated to the optic nerves by way of their sheaths. This case throws into bold relief the reality and the practical clinical importance of foci of infection in the posterior deeper accessory nasal sinuses, the ethmoid and the sphenoid sinuses, in producing perisinusitic reactions and, above all, an involvement of the optic nerves. To the study of this subject I have devoted myself.

LOW SYSTEMIC BLOOD PRESSURE AND ITS RELATION TO INTRA-OCULAR TENSION AND TO DISEASES OF THE OPTIC NERVE. DR. J. LAUBER, Warsaw, Poland.

Basing my conclusions on examinations in which the general vascular tension was compared with that of the retinal vessels and with the intra-ocular tension, I explain the pathogenesis of atrophy of the optic nerve by a disproportion between the blood pressure and the intra-ocular tension. General vascular hypertension (hypotension ?) associated with normal intra-ocular tension leads to atrophy, and this occurs also when normal systemic blood pressure is associated with increased intra-ocular tension (glaucoma). The logical treatment consists in raising the blood pressure and lowering intra-ocular tension, when there is systemic hypotony, and in lowering the ocular hypertension but avoiding lowering the general blood pressure when glaucoma is present. Antisyphilitic treatment often provokes hypotony. Careful control of systemic blood pressure in the course of antisyphilitic treatment or, in fact, the treatment of general disease of any kind is indispensable if one is to avoid the onset or the advance of atrophy of the optic nerve.

NOTE ON GLAUCOMATOUS EXCAVATION. DR. AMSLER, Lausanne, Switzerland.

No abstract was submitted.

RESULTS OF TREATING GLAUCOMATOUS STATES WITH A NEW CALCIUM-MAGNESIUM COMPOUND. DR. JEAN GALLOIS and DR. P. VIARD, Paris.

We used intravenous injections of magnesium hyposulfite and calcium chloride in an isotonic saline solution in the treatment of various forms of increased intra-ocular tension. Summing up the results after eighteen months of trial and observation, we conclude that the therapeutic results were satisfactory in simple chronic glaucoma but often negative in acute glaucoma with high tension (*grandes hypertensions*) and in hemorrhagic glaucoma. The action of this compound seems to be a combination of a primary effect of capillary vasodilatation without arteriolar dilatation and without action on the pupil, and a secondary alteration of the hydrogen ion concentration.

TRAUMATIC PARALYSIS OF BOTH NERVES OF THE SIXTH PAIR. DR. P. DUPUY-DUTEMPS, Paris.

A cranial injury was followed by coma, otorrhea, paresis of the right side of the face and bilateral paralysis of the abducens muscle. The functional disturbances disappeared in the course of several months. I consider that the symptoms were due to a compression or, more probably, a contusion of both nerves.

SYNDROME OF THE FLOOR OF THE ORBIT. DR. C. DEJEAN, Montpellier.

In two patients, aged 44 and 56 years, I observed a symptom complex corresponding to that of the development of a neoplasm having its origin in the floor of the orbit and giving rise to the following clinical signs: in the initial (incipient) stage, painful anesthesia in the domain of the second branch of the fifth nerve; in the second (established) stage, excruciating pains and anesthesia in the region supplied by the first and second divisions of the fifth nerve, diplopia, exophthalmia and enlargement of the neighboring cervical glands, probably due to metastases; in the third stage (with extension to the apex of the orbit and to the middle fossa of the skull) violent neuralgic pains over the entire region supplied by the trigeminal nerve and down toward the neck, ophthalmoplegia, anorexia, dysphagia and terminal cachexia. The symptoms of the first stage, especially the orbitofacial neuralgia, point to a compression of the superior maxillary nerve. In the second stage the growth involves the orbit and gives rise to the pains and anesthesia in the eye, the orbit, one side of the face, the brow, and one side of the skull up to the vertex. There is diplopia due to involvement of nerves or muscles in the orbit, or both, with exophthalmos. The operative conformation of a tumor originally located on the floor of the orbit, its surgical removal, histologic examination and radiography all enabled one to follow the development and progress of the neoplasm along the trunk of the superior maxillary nerve.

SYNDROME OF HYPERTENSION OF THE ORBIT AND THE OCULOCARDIAC REFLEX OF DAGUINI (DAGNINI?).

No abstract was submitted.

SPONTANEOUS OCULAR NYSTAGMUS AND NYSTAGMUS ELICITED BY CERTAIN PROCEDURES INVOLVING THE GLOBE. DR. H. TILLÉ, Paris and DR. G. FRUCHON, Paris.

It is important to differentiate clinically between spontaneous ocular nystagmus due to lesions of certain oculomotor centers or association fibers, on the one hand, and true labyrinthine nystagmus, on the other. Spontaneous vestibular nystagmus in the course of otitis media may present a definite indication for operation. The symptomatic ensemble enables one to distinguish between the two forms of nystagmus even without tests of labyrinthine function if one bears in mind the following clinical factors: the previous history and the special morphology of ocular nystagmus with the "ear marks" of rotatory (*pendiculaire*) nystagmus, irregular nystagmus and, above all, nystagmus modified by exclusion of one eye, convergence, direction of gaze, light and darkness.

Finally, a study of the results of instrumental stimulation of the vestibular structures will demonstrate their functional integrity and, by exclusion, show that the lesion must be situated above the vestibular nuclei. From the ophthalmologic point of view one has no definite knowledge of the exact anatomic localization of lesions which cause this symptom and which are located in the "supranuclear" centers or association tracts. These are summed up with reference to the publications of Bailliart (*Bull Soc. d'opht. de Paris,* February 1935, p. 66) and Sorsby (*Brit. J. Ophth.* **15:** 1 [Jan.] 1931) as follows: 1. A peripheral ocular lesion, manifest strabismus or latent strabismus producing a stimulation analogous to the caloric, rotational or galvanic stimulation tests of the labyrinth, the slow component of the movement being directed toward the excited labyrinth or eye. Roelofs (*Arch. f. Augenh.* **98:** 401 [Jan.] 1928) is of the opinion that in such cases there is a disturbance of the peripheral mechanism for neutralization of the strabismus. 2. Associated with this peripheral lesion, one infers an involvement of the upper (superior) oculomotor pathways whose internuclear connections, although well studied by Blum, are still mysterious in their topography and physiology. This applies particularly to the posterior longitudinal bundle and to the reflex, vestibular, acoustic, sensory-reflex, visual and corticonuclear paths. This aspect of the question has been studied by C. and H. Fromaget, Sauvineau and Lafon. The two pathogenic factors, the central and the peripheral, seem to be directly connected, as Sorsby believes, judging from the fact that ocular stimulation alone may let loose the appearance of nystagmus. The pathologic origin of this symptom would be supranuclear. The peripheral mechanism would be capable of accentuating an already existent spontaneous nystagmus and giving evidence of this central involvement. Ocular nystagmus is congenital, frequently associated with strabismus and diminished by its correction. The visual acuity of the eye under observation diminishes when the fellow eye is covered in those cases in which the nystagmus is accentuated by occlusion of the fellow eye. Sight is improved, on the other hand, in those cases in which occlusion of the fellow eye lessens the intensity or the extent of the nystagmus. Mental deficiency was common but by no means constant in these subjects. On the contrary, some of the subjects were, in this respect, much above normal.

Buried Treasures Discovered in Old Books on Ophthalmology. Dr. Villard, Montpellier.

No abstract was submitted.

Some Results of Subungual Capillaroscopy in Ophthalmologic Practice. Dr. H. Viallefont and Dr. R. Lafon, Montpellier.

The circulation of the blood in thirty-five patients with ocular disease was studied by the procedure mentioned in the title. While there is no direct morphologic relation between the systemic capillaries and pathologic conditions of the ocular tissues, it was noted in several morbid intra-ocular states, particularly glaucoma and certain forms of atrophy of the optic nerve, that the capillary circulation was slowed. In retinal detachment there does not seem to be any "mirroring" of a pathognomonic nature in the capillary circulation.

EXPERIMENTAL STUDY OF OCULAR INFECTIONS DUE TO BACILLUS COLI IN GUINEA-PIGS AND RABBITS. DR. R. HERRMANN and DR. P. KIEWE, Geneva, Switzerland.

No abstract was submitted.

TREATMENT OF OCULAR DISTURBANCES OF DENTAL ORIGIN. DR. L. DOR, Lyons.

No abstract was submitted.

The following papers were presented in connection with the Report on Intracapsular Extraction of Cataract at the second session on Tuesday, May 14, but no abstracts were submitted.

ADVANTAGES AND DISADVANTAGES OF TOTAL EXTRACTION OF CATARACT (Lantern Slides). DR. H. ARRUGA, Barcelona, Spain.

STATISTICS ON INTRACAPSULAR EXTRACTION. DR. GABRIEL-PIERRE SOURDILLE, Nantes.

EXTRACTION OF THE CRYSTALLINE LENS IN THE CAPSULE. DR. GILBERT SOURDILLE, Nantes.

EXTRACTION OF TRAUMATIC CATARACT WITH FORCEPS. DR. EMILIO RAVERDINO, Turin, Italy.

Cases are reported and lenses presented of a type which can in favorable cases be removed by use of the forceps, with as good results as with total extraction. The procedure is not advisable with large wounds of the anterior capsule or wounds penetrating to the vitreous. Capsule forceps with blunt spoon tips (*à cuillère*) such as Arruga's model or mine should be used.

DISCUSSION OF PAPER ON TRACHOMA BY CUENOD AND NATAF, TUNIS

DR. A. TERSON, Paris: In order to lessen the pain caused by copper sulfate, an oily collyrium should be instilled immediately after the cauterization of the conjunctiva, preferably one with a *p*-aminobenzoyl-z, 2-dimethyl-3-diethyl-aminopropanol hydrochloride base, 1:15, which at once quiets the eye and at the same time protects the cornea.

DR. M. KALT, Paris: It is important, in conjunctivitis granulosa with corneal complications and photophobia, to employ a mode of treatment which will not cause pain and which will not make one run the risk of making matters worse. Hence, I am afraid of the copper stick. I have remained true to silver nitrate, instilling a 1 per cent solution repeatedly after cocainization and following up with cocaine again to neutralize the caustic action of the silver nitrate. Atropine is then dropped into the eye. This treatment can be applied twice daily. In my practice it has proved to be most efficacious.

NEW METHOD OF DETERMINING WHEN RAYS FROM A MAGNIFYING LENS ARE PARALLEL, CONVERGENT OR DIVERGENT. DR. LÓPEZ-ENRÍQUEZ, Madrid Spain.

The magnification of a loupe is invariable when the observation is made with parallel rays. But when the object does not coincide exactly with the focal plane, the enlargement of the image is variable, according to the distance of the observer's eye from the lens. If the object is beyond the focal distance of the lens (converging rays) the image increases in size as one withdraws the system from the eye. If the object is nearer to the loupe than its focal plane (diverging rays) the image becomes smaller. The apparatus for these experiments consists of an optical bench moved by hand along a horizontal scale without the distance between the different systems being changed. The images are measured, in binocular projection, by being thrown on a screen and compared.

Book Reviews

Die Störungen des Lichtreflexes der Pupille bei den luetischen Erkrankungen des Zentralnervensystems. Beiträge zur Frühdiagnostik der Lues nervosa. By Otto Löwenstein, M.D., one time professor and director of the pathopsychological institute of Bonn University, and directing physician of the Rhenish provincial children's hospital for the mentally abnormal in Bonn; at present in Nyon, Switzerland, La Métairie Clinic. Price, 5 francs; 4 marks. Pp. 92, with 31 photographs, diagrams and charts. Basel: Benno Schwabe & Company, 1935.

This book is based on the study of one hundred patients presenting syphilitic disorders of the central nervous system, who were examined and followed clinically during a period of ten years.

Since the ordinary methods of examination do not bring out the details of pupillary reaction, Löwenstein refined on the observation and the recording of these reactions by making cinematic photographs of the two pupils before, during and after exposure of one eye to light. His procedure is as follows: The eyes are illuminated continuously from each side by light which has passed through a blue filter. A motion picture camera run by a motor and giving fourteen exposures to the second is started, while the patient fixes a distant object. A light of several thousand candle power is then flashed on one eye for a second, while the photography continues. The film so obtained is projected on a screen, and the width of the contracting and dilating pupil is plotted with protractors on a strip of paper ruled vertically for seconds and horizontally for millimeters. By this means a down and up curve is obtained which is a graphic record of the extent and the rapidity of the contraction and subsequent dilatation of each pupil after one pupil is exposed to light.

Following the account of his method, the author takes up the description of the curves charted for normal eyes. There is first a brief period of latency, then a down curve which is notched, followed by an up curve also notched, because the course of the contraction and dilatation is broken and not uniformly continuous. He found four normal types: (1) prompt contraction, prompt dilatation; (2) prompt contraction, slow dilatation; (3) slow contraction, prompt dilatation; (4) slow contraction, slow dilatation.

When the flash of light is repeated a number of times, fatigue of the light reflex comes on, and the curve of contraction and dilatation gradually becomes more shallow, until at length the flash may cause even dilatation of the pupil—the paradoxical light reflex.

Psychic factors produce dilatation of the pupil, and if, for example, the patient is frightened or is in pain, his light reflex is reduced.

The plotted curve for the direct reflex is represented by a continuous line and that for the consensual reflex by a dotted line on the same strip of paper. With normal eyes these two lines are nearly alike, and when there is a marked difference between the two, one is dealing with a pathologic condition.

The second half of the book is concerned with the symptomatology of abnormal reflexes in syphilitic disorders of the central nervous system. In general, there is great diversity of these abnormal reflexes. They may be slight, even in advanced dementia paralytica, and, on the contrary, they may be well marked even at an early stage when no other syphilitic symptoms can be elicited. In this period the pupillary disturbances are of great diagnostic significance, and it is precisely in these early stages of syphilis that treatment is most successful.

The symptomatology is discussed under several captions: (1) abnormally fatigable light reflexes, (2) changes in the tempo of the contraction and of the subsequent dilatation, (3) abnormal forms of consensual reflex, (4) lengthening of the period of latency before contraction occurs and also lengthening of the period before dilatation ensues and (5) paradoxical dilatation of the pupil on exposure to light.

Here Löwenstein describes and charts about forty symptoms and groups of symptoms with curves departing from the normal, which are of value in diagnosis. Lack of space prevents even the enumeration of these many varieties of palpably abnormal curves.

Formerly it was believed that marked sluggishness of the light reflex was an indication of syphilis, but in the last two or three decades it has been recognized that sluggish pupils are found in other morbid conditions, particularly dementia praecox and epidemic encephalitis. And not the least of Löwenstein's important contributions to the present knowledge of pupillary reflexes lies in his showing how one may distinguish the syphilitic from the nonsyphilitic disturbances.

Finally, it may be said that the author has devised a new method of precise physiologic measurement which is comparable with the measurements obtained by the use of the perimeter and the tonometer.

WARD A. HOLDEN.

Ueber die primären Tumoren des Sehnerven und der Sehnervenkreuzung. By Åke Lundberg. Inaugural dissertation, Karolinisches Institut, Stockholm. Pp. 164, with 28 illustrations. Stockholm: Nordiska Bokhandeln, 1935.

In this monograph Lundberg deals with the clinical and pathologic aspects of primary tumors of the optic nerve and chiasm. In tabular form are listed the important features of the one hundred and fourteen cases of this condition the reports of which were published between 1912 and 1934, thus bringing to date Hudson's exhaustive summary. To this series are added ten cases of the author's. These too are described clinically and histologically, the microscopic specimens being stained with the modern silver stains introduced by the Spanish school of histologists. In nine cases the tumors were gliomas of the type designated as oligodendrocytoma by Río Hortega. Five of these involved the intra-orbital portion of the optic nerve, four the chiasmal region. The tenth case was one of meningioma, in which the tumor occupied the region of the inner opening of the optic canal.

There is a brief section devoted to clinical diagnosis. The age incidence of gliomas is earlier than that of meningiomas and in the former loss of vision usually precedes the exophthalmos, whereas in meningiomas the exophthalmos is apt to come first. The meningiomas are also more

apt to cause great limitation of motility. The roentgen rays are of most use in those cases in which a decided increase in the diameter of one optic canal indicates an extension of the tumor of the nerve intracranially. Both types of tumor are relatively benign; i. e., they grow slowly and do not usually break through the nerve sheaths to metastasize. The real danger that they will extend along the nerve intracranially constitutes a clear indication for their earliest possible surgical removal.

The monograph ends with a section devoted to the investigation of the neuroglial content of the optic nerve and chiasm in embryos of various sizes. A bibliography is included.

O. P. PERKINS.

L'Extraction capsulo-lenticulaire de la cataracte. Report Presented to the French Ophthalmological Society, May 14, 1935. By R. De Saint-Martin, Toulouse. Pp. 475, with 76 illustrations and 15 color plates. Paris: Masson & Cie, 1935.

This truly monumental work by the Toulouse eye surgeon is, as indicated in the title, a report on the complete or intracapsular removal of the cataractous lens. But it is much more. With a thoroughness and attention to detail which one is wont to characterize traditionally as typical of the *Arbeit* of German scholars, the author has given not only a complete account of the history, development and present status of the capsulo-lenticular operation but a critique with statistics, from his own practice and that of many other surgeons, of the various procedures of antisepsis, akinesia and anesthesia, the various steps, the different methods of preparation for operation and their results. The mechanical procedures on which total extraction is based, from the early, simple expression of Smith of Jullundur and Amritsar to the vacuum or suction operations, the extraction with capsule forceps and, the latest development, adhesion extraction by diathermy-coagulation, are discussed in detail as to construction of apparatus, the technic of manipulation, the operative results, sequelae and complications. There are chapters on the histologic changes observed after these surgical procedures and other chapters on morbid clinical manifestations and their dependence on various factors of operative technic. The data of slit-lamp and ophthalmoscopic examination of the eye some time after operation are presented. The comprehensive nature of the topics discussed, the wide scope of the work, the clear and intelligent style and manner of presenting a wealth of detail, the instructive object lessons offered by a large number of beautiful colored plates, and, last but by no means least, a bibliography including the years from 1740 to 1934 and part of 1935, make this work an authoritative guide to the surgeon and clinician and, above all, an invaluable reference hand-book for the ophthalmologist, be he student or teacher.

PERCY FRIDENBERG.

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President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.
Place: Cairo. Time: 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. A. J. Ballantyne, 11, Sandyford Pl., Glasgow, C 3.
Secretary: Dr. J. Ringland Anderson, 108 Collins St., Melbourne.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. R. Foster Moore, 53, Harley St., London, W. I.
Secretary: Miss Ida Mann, 7, Wimpole St., London, W. I.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. John Green, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Kansas City, Mo.: Time: May 11-15, 1936.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: New York. Time: October 1936.

American Ophthalmological Society

President: Dr. L. S. Greene, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 1-3, 1936.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston, Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. H. G. Merrill, 3245 4th St., San Diego, Calif.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Del Monte, Calif. Time: Spring, 1936.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. W. R. Fringer, 228 S. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

Southwestern Michigan Triological Society

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. George B. Garlick, 144 Golden Hill St., Bridgeport.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.
Place: Savannah. Time: May 12-15, 1936.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.
Place: Martinsville. Time: April 8, 1936.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. W. Dean, 532, 1st Ave., Council Bluffs.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. E. Binger, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Place: St. Paul. Time: May 1936.

Montana Academy of Oto-Ophthalmology

President: Dr. George A. Lewis, Roundup.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Billings. Time: February 1936.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Secretary: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.
Place: Jamestown. Time: May 1936.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Emory Hill, 501 E. Franklin St., Richmond.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
Place: Richmond. Time: May 1936.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Herschel Crawford, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Stacy C. Howell, 384 Peachtree St., N. E., Atlanta, Ga.
Place: Academy of Medicine, 13 Queen's Park. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

Chicago Ophthalmological Society

President: Dr. Robert B. Blue, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. F. W. Thomas, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Newton, Mercantile Bldg., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months. September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde E. Harner, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul T. Southgate, 704 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Walter R. Crane, 756 S. Broadway, Los Angeles
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. E. A. Leggett, 614 Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. E. Craddock, 604 Breslin Bldg., Louisville, Ky.
Place: Jewish Hospital. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. J. J. Brook, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member in alphabetical order.
Secretary: Dr. C. W. Rucker, 527 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. W. J. Blackburn, 663 Reibold Bldg., Dayton, O.
Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Kate S. Zerfoss, 119, 7th Ave., N., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St.,, New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John H. Judd, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassidy, 107 S. 17th St., Omaha
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGER OPHTHALMOLOGICAL CLUB

President: Dr. S. T. Hubbard, 294 State St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, 500 Penn Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Samuel M. Cottrell, 116 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Meyer Wiener, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. D. Brazeau, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. J. B. Stone, 1105 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Arnold B. Kauffman, 104 W. Onondaga St., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St. W., Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.